7.2.6.4.4 ARA-12, ARA-III Radioactive Waste Leach Pond—Site ARA-12 is an unlined surface impoundment in a natural depression that was active from 1958 to 1991. The 5,748-m^2 (61,870-ft^2 or 1.4-acre) site is unfenced, and the vegetation at the site includes grasses, junipers, and willows. The pond is usually dry but holds water on an intermittent basis during periods of high precipitation. Contaminants at ARA-12 are radionuclides and metals in surface and subsurface soil. It should be noted that detection limits for samples collected at this site were above EBSLs for some organic contaminants. Contaminants of potential concern at ARA-12 are metals (including mercury) and benzo(a)pyrene.

7.2.6.4.5 ARA-16, ARA-I Radionuclide Tank (ARA-729)—Site ARA-16 is a 1,000-gal underground storage tank within a concrete vault, covered by 1.1 m (3.5 ft) of soil. The site encompasses only about 61 m^2 (660 ft^2). The tank had received radioactive liquid waste, VOCs, chlorinated paraffin, and mixed acids from 1959 to 1988. The tank was partially excavated in 1988 during the shutdown procedure. Soils from the excavation were replaced over the tank after shutdown. Soil contaminants at ARA-16 include radionuclides, organics, and metals. Fluoride is the only COPC retained for analysis in the ERA.

7.2.6.4.6 ARA-25, ARA-I Soil Beneath the ARA-626 Hot Cells—Site ARA-25 comprises contaminated soils discovered beneath the floor slabs in the two hot cells (Hot Cells No. 1 and No. 2) in building ARA-626. Floor drains and accompanying drain lines in the hot cells were at one time connected to the ARA-729 radionuclide tank (site ARA--16). The ARA-729 tank contains PCB-contaminated, listed mixed waste and transuranic radionuclides. Soil samples collected beneath concrete slabs removed during D&D operations indicate radionuclide, metal and organic contamination in surface soil. Because sampling was limited to the surface of the soil, the ERA incorporated the assumption that maximum detected concentrations extend from the surface down to the basalt interface at a depth of 5 ft. The concrete floor slabs (and the associated fixed contamination) will be removed and are not addressed in the risk assessment.

7.2.6.4.7 PBF-04, PBF Control Area Oil Tank at PBF-608 Substation Outside PBF Fence—Site PBF-04 is the former site of a 1,000-gal underground storage tank that contained heating fuel for the PBF Substation Control House. The tank and some contaminated soils were removed in 1990, and the excavation was backfilled with clean soil. The 11-m^2 (122-ft^2) site is gravel-covered and is located inside the substation containment fence. The area is surrounded by native sagebrush community. Xylene in subsurface soil is the only COPC at PBF-04.

7.2.6.4.8 PBF-10, PBF Reactor Area Evaporation Pond (PBF-733)—Site PBF-10 is the site of a 1,820-m^2 (19,600-ft^2) lined surface impoundment that received effluent from 1972 to 1984. These effluents included chromium-contaminated coolant water and demineralizer system discharges containing resins, sulfuric acid, and sodium hydroxide. Portions of the pond were remediated in 1994, and, in 1995, the liner was removed. The pond berm was bulldozed, and the area was graded and seeded with native grasses. Radionuclides and chromium were detected in soils sampled at PBF-10. Chromium is the only COPC retained for the ERA.

7.2.6.4.9 PBF-16, PBF SPERT-II Leach Pond—This site is a fenced, unlined surface impoundment that was used for disposal of demineralizer effluent, water softener waste, and discharges from drains in reactor building from 1959 to 1964. The site is approximately 3570 m^2 (38,400 ft^2). Metals are the contaminants in soil at PBF-16. Lead and mercury are the COPCs retained for the ERA.

7.2.6.4.10 PBF-21, SPERT-III Large Leach Pond—This pond received waste from the sump pump in the SPERT-III reactor building from 1958 to 1968. The pond was backfilled with clean fill, leveled, and seeded with native vegetation in 1983 as part of a D&D program. The 288-m^2

(3,099-ft^2) site is surrounded by the native sagebrush community. Contaminants of potential concern include cobalt and copper in subsurface soil.

7.2.6.4.11 PBF-22, SPERT-IV Leach Pond (PBF-758)—Site PBF-22 was the site of an unlined impoundment that received effluent from the SPERT-IV reactor from 1961 to 1970. In the early 1980s, the pond received contaminated primary coolant effluent from the PBF reactor. In 1985, six 1.7-m^3 (64-ft^3) boxes of contaminated soil were removed from the pond. The vegetation at the 5,008-m^2 (53,908-ft^2 or 1.2-acre) site currently includes tall sagebrush, rabbitbrush, and grasses. Contaminants of potential concern include PCBs and metals in surface and subsurface soils.

7.2.6.4.12 PBF-26, PBF SPERT-IV Lake—This site is a 20,092-m^2 (216,276-ft^2 or almost 5-acre) unlined surface impoundment formerly used for discharge of reactor secondary cooling water. Site PBF-26 is located next to PBF-22. In 1992, all discharges to the PBF-26 impoundment were ended. The site has been revegetated with crested wheatgrass and is adjacent to tall sagebrush and basalt outcrop communities. Contaminants of potential concern at PBF-26 are PCBs and metals in surface and subsurface soils.

7.2.7 Pathways of Contaminant Migration and Exposure

The potential risk posed by contaminants in surface and shallow subsurface soil at WAG 5 was evaluated by developing models to determine exposure pathways and receptors, and using bioenergetics-based exposure models to estimate exposure. However, an assumption that no pathway to ecological receptors exists for this medium was incorporated into the WAG 5 ERA. Groundwater is considered inaccessible to ecological receptors because of the depth to the aquifer at the INEEL (60 to 180 m [200 to 900 ft]) and the large distance to surface springs (more than 160 km [100 mi.]) (EG&G 1993). Waste Area Group 5 soil COPCs include metals and organic compounds. All radionuclides were eliminated in the earlier EBSL and background comparisons.

7.2.7.1 Surface Soil. Contaminated surface soil represents the major source of possible contaminant exposure for WAG 5 ecological receptors. Surface soil, as defined for use in INEEL WAG ERAs, includes the uppermost 0.15 m (0.5 ft) of soil. Seven of the 12 WAG 5 sites of ecological concern represent sources of surface soil contamination resulting from past contamination.

The model for ecological pathways and exposure to WAG 5 contaminated surface soil is shown in Figure 7-4. The model depicts the various mechanisms for surface soil contamination transport as follows:

- Wind and water erosion
- Leaching and infiltration
- Plant uptake
- Burrowing animal translocation.

Transportation of contaminated soils through these mechanisms may result in contamination of various other media or secondary sources, including the following onsite and offsite sources:

- Surface water
- Surface soil

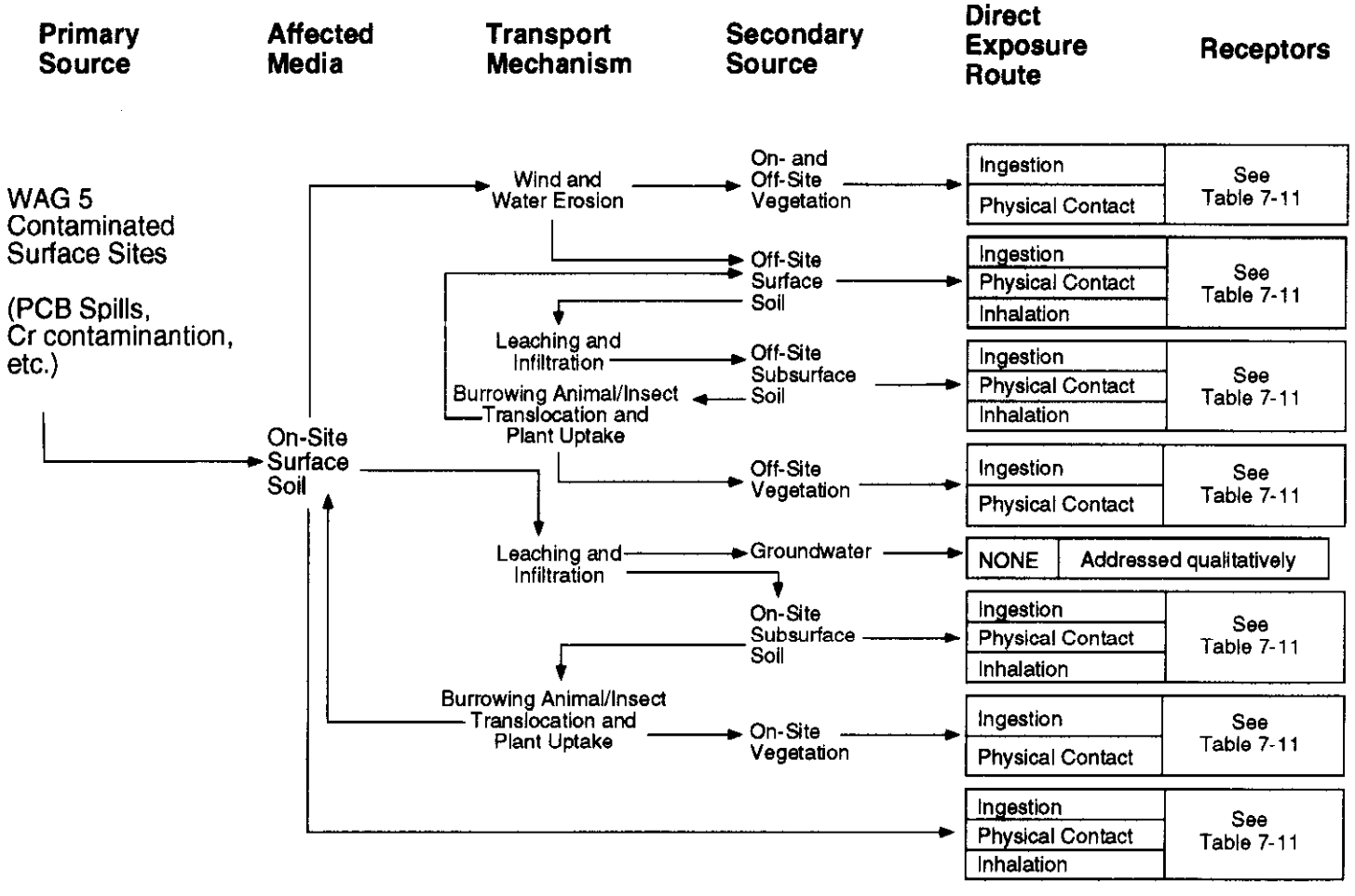

Figure 7-4. Model for ecological pathways and exposure for WAG 5 surface contamination.

- Subsurface soil
- Vegetation.

Receptors having the potential for direct exposure to WAG 5 surface soils are presented in Table 7-11. Ecological receptors can be exposed to contaminated media directly through ingestion of contaminated vegetation, water, and prey; incidental ingestion of soil; or through physical contact or inhalation. Inhalation and physical contact, however, are considered to play minor roles in the exposure to surface contamination for WAG 5 and are not evaluated in this assessment. The functional groups identified as having direct exposure include most terrestrial avian, mammalian, reptilian, and insect species potentially present in the WAG 5 area.

Table 7-11. Summary of WAG 5 exposure media and ingestion routes for INEEL functional groups.

Receptor	Surface Soils	Subsurface Soils	Vegetation	Sediments	Prey Consumption		
					Invertebrates	Mammals	Birds
Avian herbivores (AV122)	X						
Avian insectivores (AV210A)				X	X		
Avian insectivores (AV222)	X				X		
Avian insectivores (AV232)				X	X		
Avian carnivores (AV310)	X					X	X
Northern goshawk	X					X	X
Peregrine falcon	X					X	
Avian carnivores (AV322)						X	
Bald eagle						X	
Ferruginous hawk						X	
Loggerhead shrike						X	X
Avian carnivores (AV322A) Burrowing owl	X	X			X	X	
Avian omnivores (AV422)			X		X	X	X
Mammalian herbivores (M122)	X		X				
Mammalian herbivores (M122A)	X	X	X				
Pygmy rabbit	X	X	X				
Mammalian insectivores (M210A)	X				X		
Townsend's western big-eared bat	X				X		
Small-footed myotis	X				X		
Long-eared myotis					X		
Mammalian insectivores (M222) Merriam's shrew	X			X	X		
Mammalian carnivore (M322)	X					X	
Mammalian omnivores (M422)	X	X	X		X		
Reptilian carnivores (R322)						X	
Plants							

7.2.7.2 Subsurface Soil. The model for ecological pathways and exposure for WAG 5 contaminated subsurface soils is presented in Figure 7-5. Several of the WAG 5 sites of concern are contaminated subsurface soil sites resulting from buried contaminated soil or sediments, leaking underground storage tanks, and past surface spills followed by leaching. At Sites ARA-02, ARA-03, PBF-04, PBF-10, and PBF-21, only subsurface soil contaminants have been detected. Contaminants have been detected in both surface and subsurface soil at the ARA-01, ARA-12, and PBF-22 sites. For the WAG 5 ERA, subsurface soils are defined at depths of 15 cm to 3 m (0.5 to 10 ft). Contaminants in subsurface soil can be transported to ecological receptors by plant uptake and translocation by burrowing animals. Contamination at depths greater than 3 m (10 ft) are considered inaccessible to ecological receptors because that depth generally is below the root zone of plants and the burrowing depth of ground-dwelling animals.

Insects and burrowing animals have the potential for bringing contaminated subsurface soils and buried waste to the surface. Once contaminated soil is brought close to the surface, transport and exposure scenarios for ecological receptors are the same as for surface soil. For subsurface contamination, inhalation and direct contact (by burrowing animals) are more important exposure routes than for surface contamination. Receptors having a potential for direct exposure to WAG 5 subsurface soil contamination are presented in Table 7-11. These receptors include burrowing animals and deep-rooting plants. Because subsurface soil contamination may be translocated to the surface by burrowing animals and plant uptake, other terrestrial species also have some potential for exposure through this pathway. However, no site-specific or other data were researched to confirm or evaluate this potential source of surface contamination, which is considered a data gap. A thorough literature analysis of this potential contamination exposure route should be evaluated in the INEEL-wide (OU 10-04) ERA.

7.2.8 Conceptual Site Model

The models for pathways and exposure for surface and subsurface soil were integrated to produce the WAG 5 CSM shown in Figure 7-6. The CSM reflects both direct and indirect (i.e., predation) receptor exposure pathways for WAG 5 COPCs.

7.2.9 Development of Assessment Endpoints

This section addresses the development of assessment endpoints, which are "formal expressions of the actual environmental values that are to be protected" (Suter 1989). Assessment endpoints developed for the WAG 5 ERA are presented in Table 7-12. The endpoints were developed around the protection of INEEL biota represented by functional groups and individual T/E and sensitive species known to exist at WAG 5 and identified as having potential for exposure to COPCs. Each T/E species is addressed individually in the risk analysis, whereas potential effects on other receptors of concern are evaluated at the functional group level (see Section 7.2.4.2). Assessment endpoints defined for the WAG 5 ERA reflect the INEEL-wide hazard control and policy goals discussed in the INEEL ERA guidance manual (VanHorn, Hampton, and Morris 1995) and incorporate the suggested criteria for developing assessment endpoints including ecological relevance and policy goals (EPA 1992; Suter 1993).

Assessment endpoints are the focus of WAG ERA risk characterization and link the measurement endpoints to the WAG ERA goals. The primary objective of the WAG 5 ERA is to identify COPCs and the levels of those contaminants that represent potential risk to WAG 5 ecological components. Though adverse effects caused by physical stressors also are of concern in evaluating potential risks to INEEL ecological components, these effects are not addressed by the WAG 5 ERA. An approach using hazard quotients (HQs) was employed to establish the potential for contaminants to contribute to ecological risk to WAG 5 individuals and populations. Hazard quotients are used to indicate whether a potential exists for adverse effects. The use of HQs as indicators of adverse effects is discussed in detail in Section 7.4.1.

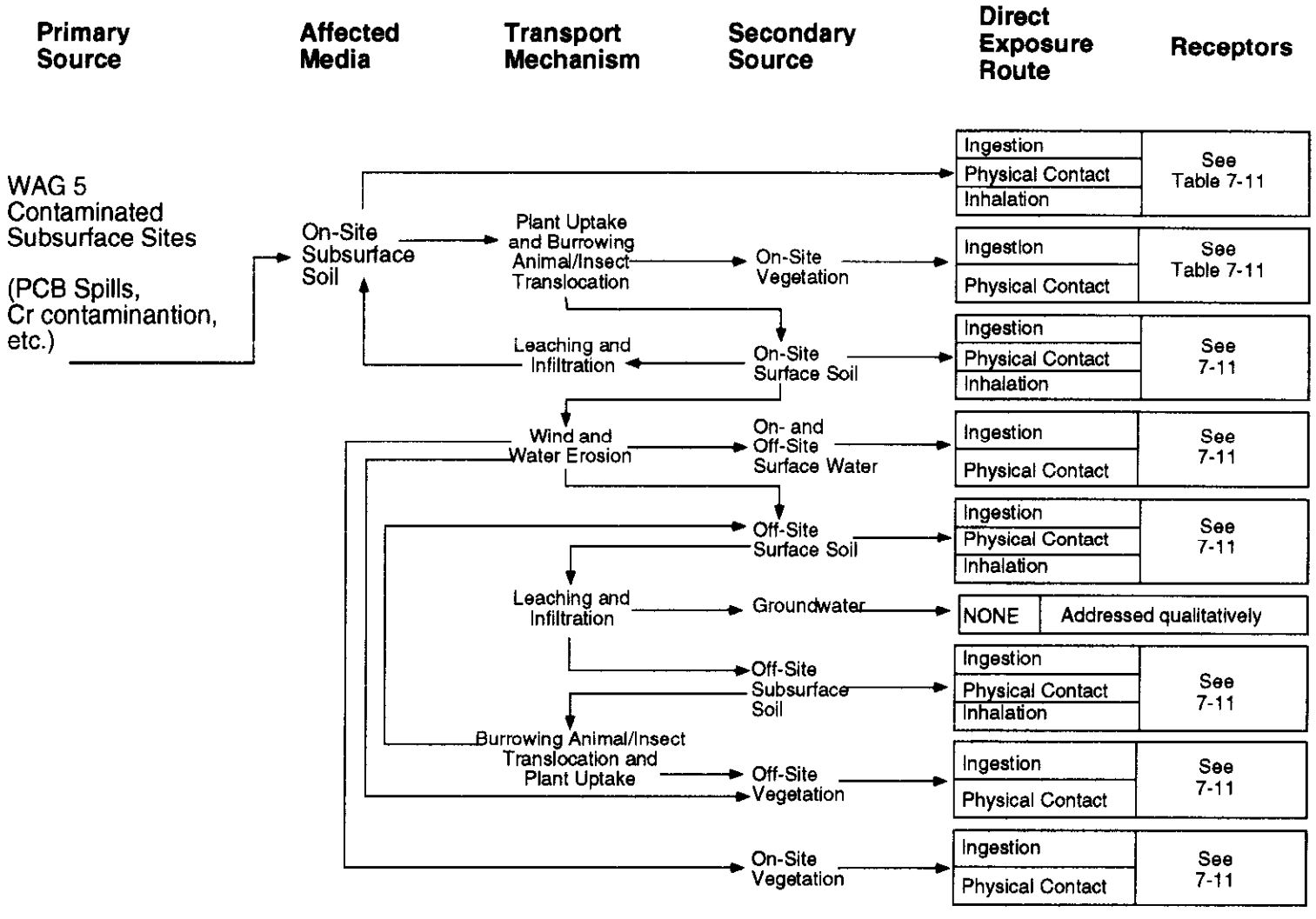

Figure 7-5. Model for ecological pathways and exposures for WAG 5 subsurface contamination.

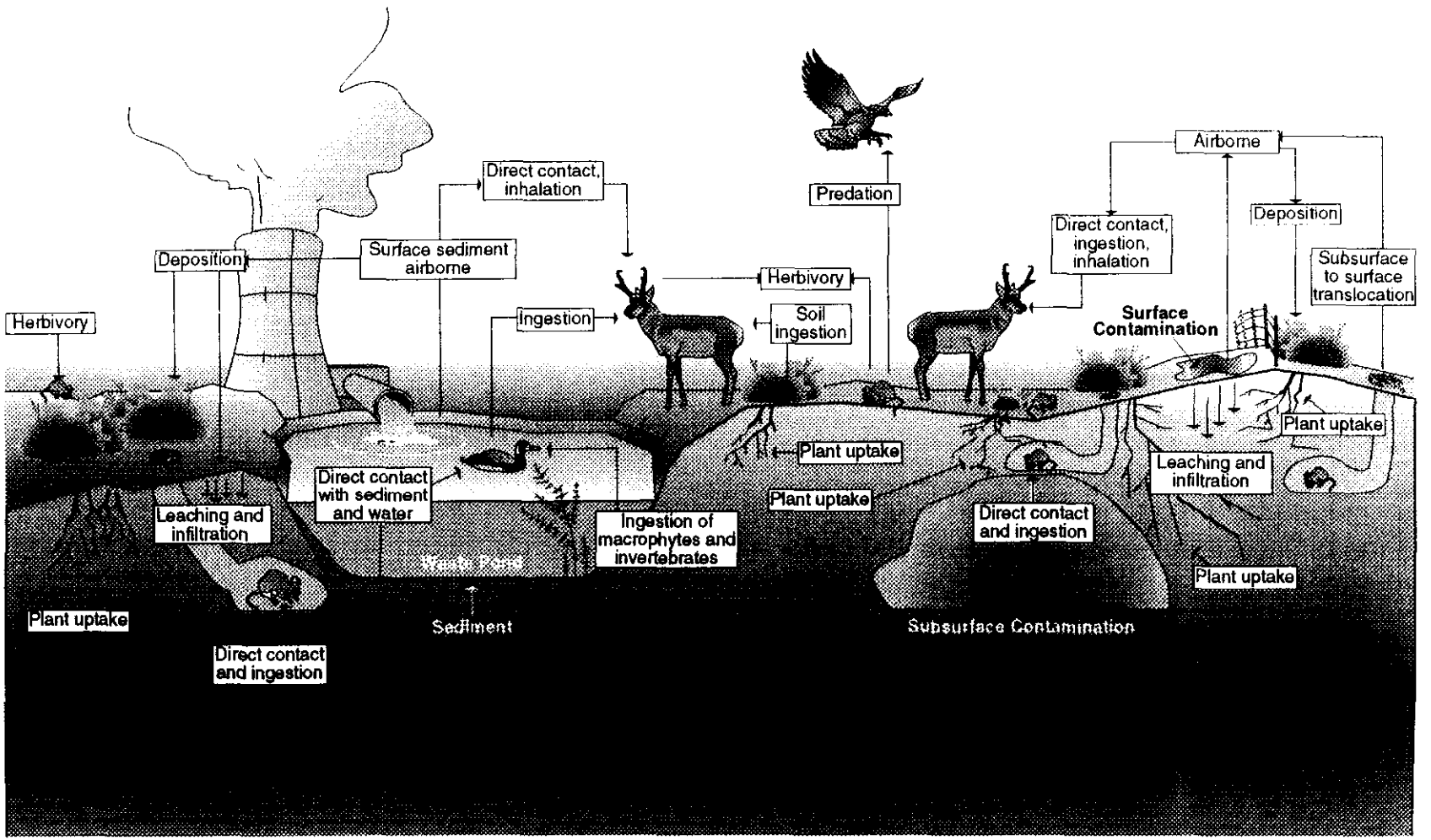

Figure 7-6. Ecological conceptual site model for WAG 5.

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Table 7-12. Summary of assessment endpoints for WAG 5 ERA (Suter 1993).

Management Goals	WAG ERA Endpoint	Indicator of Risk[a]
Maintain INEEL threatened or endangered T/E and Category 2 (C2) (now referred to as species of concern) individuals and populations by limiting exposure to organic, inorganic, and radionuclide contamination.	No indication of possible effects to T/E and C2 wildlife and plants as a result of contaminant exposure.	HQ[b] ≥ target value
Maintain INEEL T/E and C2 individuals and populations by limiting exposure to physical stressors.	Not addressed by WAG ERA	N/A
Maintain survival, abundance and diversity of INEEL native biota by limiting exposure to organic, inorganic, and radionuclide contamination.	No indication of possible effects to WAG native vegetation communities as a result of contaminant exposure.	HQ ≥ target value
	No indication of possible effects to WAG wildlife populations as a result of contaminant exposure (represented by functional groups identified in the site conceptual model: small mammals, large mammals, songbirds, raptors, top predators, and invertebrates).	HQ ≥ target value
Maintain survival, abundance, and diversity of INEEL native biota by limiting exposure to physical stressors.	Not addressed by WAG ERA	N/A

a. Based on original guidance provided by EPA (1992), this column might have been called the "measurement endpoint." Subsequent guidance from EPA (1996) now discusses measures or indicators of effects.

b. HQ = hazard quotient. The target value is 1 for nonradionuclide contaminants and 0.1 for radionuclide contaminants. The HQ approach does not consider variability and uncertainty in either exposure or toxicity estimates and, therefore, does not represent a statistical probability of occurrence of adverse ecological effects. Hazard quotients essentially provide a "yes" or "no" determination of risk and are, therefore, well suited for screening-level assessments (EPA 1988b). A limitation of the quotient method is that is does not predict the degree of risk or magnitude of effects associated with specified levels of contamination (EPA 1988b).

7.2.8 Measurement Endpoint Selection

This section describes the selection of measurement endpoints for the WAG 5 ERA. Measurement endpoints are measurable responses of the exposure of ecological receptors to contaminants that can be related to WAG ERA assessment endpoints. For the WAG 5 ERA, the ecological components (flora and fauna) were not measured or surveyed directly. Rather, published references were used as the primary sources of ecological and toxicological data from which measurement endpoints were derived. Values

extracted from these references were used to calculate EBSLs for all ecological receptors and to develop toxicity reference values (TRVs) for the COPCs. Table 7-13 summarizes the measurement endpoints developed to address WAG 5 assessment endpoints. Quantified critical exposure (QCE) levels and adjustment factors (AFs) were constructed from the literature to develop appropriate TRVs for receptors associated with WAG 5 contaminant pathways. Criteria for development of these TRVs are discussed in Section 7.3.3.1. In general, the criteria incorporate the requirements for appropriate measurement endpoints, including relevance to an assessment endpoint, applicability to the route of exposure, use of existing data, and consideration of scale (VanHorn, Hampton, and Morris 1995).

Published values for species dietary habits, home ranges, site use, exposure duration, soil ingestion, food digestion, and body weights for the representative species and the contaminant exposure-point concentrations in each medium were used to calculate dose for each affected receptor (see Section 7.3.2).

A measurement endpoint is the modeled dose as compared to the TRV for each contaminant for each receptor or functional group. The modeled dose was divided by the TRV to produce an HQ for each contaminant and receptor of concern. Hazard quotients are used to measure whether assessment endpoints have been attained—that is, whether survival and reproductive success are ensured for the receptor groups being assessed (i.e., the HQs are less than 1 for all receptors for each contaminant).

7.3 Analysis

The risk analysis step of the WAG 5 ERA involves assessing exposure to contaminants (characterization of exposure) and the potential effects of exposure (characterization of effects). The two assessment activities are conducted interactively to ensure that the methods used to assess exposure and its effects are compatible. Assessing exposure and its effects is based on the ecological endpoints and conceptual models derived during the problem formulation phase of the assessment (Section 7.2).

A primary step in analyzing risk is to estimate the magnitude, frequency, duration, and route of exposure to site-related contaminants by ecological receptors. Accomplishing this task involves completing the following steps:

1. Research and discuss the factors that influence contaminant fate and transport

2. Estimate dose for all functional groups and contaminants.

7.3.1 Discussion of Contaminant Fate and Transport Properties

The behavior and fate of the contaminants in the terrestrial environment are discussed in this section. Environmental fate properties are important because they provide information on the environmental behavior and, thereby, the bioavailability, of contaminant compounds throughout various environmental media. No formal fate and transport modeling was conducted for the WAG 5 ERA. Therefore, information on fate and transport properties was obtained from the scientific literature. All radionuclides for ARA-25 were eliminated in the preliminary screening presented in Section 7.2.6.3. The nonradionuclide contaminants for WAG 5 surface and subsurface soils that were evaluated in the WAG 5 ERA, include the following:

Table 7-13. Summary of WAG 5 ERA endpoints.

WAG 5 Assessment Endpoint	Ecological Component	Functional Group (Other Groups Represented)	Measurement Species (Toxicity Reference Value Test Species)
No indication of possible effects on T/E and C2 individuals and populations as a result of contaminant exposure.	Pygmy rabbit	M122A (M123)	Rat, mouse/meadow vole (M122A), and deer mouse (M422)
	Peregrine falcon, and northern goshawk	AV310	Chicken, goshawk, and American kestrel/red-tailed hawk (AV322)
	Ferruginous hawk, loggerhead shrike, bald eagle, and burrowing owl	AV322, AV322A	Chicken, goshawk, and American kestrel/red-tailed hawk (AV322)
	Sagebrush lizard	R222	None located
No indication of possible effects on WAG 5 native vegetation communities as a result of contaminant exposure.	Vegetation	Sagebrush and bunchgrass	Bush beans and crop plants
No indication of possible effects on WAG 5 wildlife populations as a result of contaminant exposure (represented by functional groups identified in the site conceptual model: small mammals, large mammals, song birds, raptors, and top predators, invertebrates).	Small mammals	M422, M122A (M222, M123)	Rat, mouse/meadow vole (M122A), and deer mouse (M422)
	Mammalian carnivores and omnivores	M422A, M322	Rat, mouse, dog, cat, and mink/fox
	Mammalian herbivores	M121, M122, M122A	Rat, mouse, and mule deer/pronghorn
	Avian carnivores	AV322, AV322A, M122A	Goshawk (AV310) and American kestrel/red-tailed hawk (AV322)
	Avian herbivores	AV121, AV122	Chicken, pheasant, quail, and passerines/sharp-tailed and ruffed grouse
	Avian insectivore	AV210, AV222 (AV210A, AV221, AV22A)	Chicken, pheasant, quail, passerines/American robin (AV222), and cliff swallow (AV210A)
	Avian omnivores	AV422	Chicken, pheasant, turkey, black, mallard
	Mammalian insectivore	M210A (M210)	None located
	Reptiles	R222, R322	Western racer (none located)
	Invertebrates	Phytophagous, saprophagous, and entomophagous	Unidentified

- Antimony
- Aroclor-1248
- Aroclor-1254
- Arsenic
- Barium
- Benzo(a)pyrene
- Cadmium
- Chromium
- Cobalt
- Copper
- Fluoride
- Lead
- Manganese
- Mercury
- Nickel
- Selenium
- Silver
- Thallium
- Vanadium
- Xylene
- Zinc.

Many of the WAG 5 contaminants are metals. Soils represent the most concentrated source of metals in the terrestrial environment. Particulate matter readily sorbs metals, which may complex with various anions such as carbonates and sulfides and thus modify their water solubility. Such sorption and complexation (typically) diminishes the bioavailability of metals in soils and sediments or aqueous systems (Adams, Kimberle, and Barnett 1992).

The health risks posed by trace metals in soils are not determined solely by their quantity. A number of contaminant, environmental, and biological conditions and processes influence the accessibility and availability of metals to organisms and, hence, the toxicological significance of the metals. First, speciation is a major determinant of the fate, bioavailability, absorption, and toxicological characteristics of metal compounds. Second, the distribution coefficient between soil and water (K_d) depends on both the properties of the metal and the composition of the soil. This coefficient also governs the bioavailability of a metal to organisms contacting the soil. Weakly bound metals are highly bioavailable, and more strongly bound metals are less bioavailable. Other influential factors include (1) the characteristics of the interface (e.g., lung, skin, and intestine), (2) the reactivity of the metal with the interface, and (3) the concurrent presence of other metals or other substances that may stimulate or inhibit metal uptake.

Specific factors that influence the fate and transport of the WAG 5 COPCs are presented in Sections 7.3.4 and 7.3.5, along with discussions of the ecotoxicological effects of and derivation of TRVs for these contaminants.

7.3.2 Determining Exposure

Exposures for each functional group, T/E species, and sensitive species were estimated based on site-specific life history and, when possible, feeding habits. Quantification of group and individual exposures incorporated species-specific numerical exposure factors including body weight, ingestion rate, and the fraction of diet composed of vegetation or prey and soil consumed from the affected area. Parameters used to model contaminant intakes by the functional groups are presented in Table 7-14. These values were derived from a combination of parameters that produced the most conservative overall exposure for the group. Parameter values and associated information sources are discussed in further detail in Appendix I.

The diet of each receptor was assumed to be composed of one or two food types (i.e., either or both prey and vegetation) to simplify exposure calculations. For example, herbivorous animals are assumed to

consume solely contaminated vegetation taken from the WAG 5 area. Vegetation is not broken into seeds versus vegetative parts to account for the potential differences in plant part uptake. Though warranted, breaking down the diet of individual species within a function group in more detail is beyond the scope of a WAG ERA. Most terrestrial receptors incidentally or directly ingest soil and the percent of soil ingested from that affected area also was estimated.

Exposure estimates were adjusted for the WAG 5 site areas by the use of site use factors (SUFs). The SUF is the site area (hectare [ha]) divided by the species' home range (ha) to a maximum value of 1. Home ranges for the functional groups at WAG 5 are summarized in Table 7-14. Because SUFs are calculated for each site and receptor combination, space will not allow inclusion of those values on Table 7-14. The SUFs for individual receptors can be calculated by dividing the receptor home range found on Table 7-14 by site sizes given on Table 7-18. For species with unknown home ranges, the SUF is defaulted to a value of 1. An SUF of less than 1 indicates that the home range is larger than the area affected, and it is likely that these functional groups or T/E species consume prey, vegetation, and soil from offsite areas as well.

Exposure duration (ED) is based on the migratory pattern of the receptors. The ED is determined using the status and abundance data compiled for site species (VanHorn, Hampton, and Morris 1995). Five status and abundance categories are represented: resident, breeding, summer visitor, migratory, and winter visitor. For year-round residents, the ED is assumed to be 1 (i.e., receptors potentially spend up to 100% of the year on the assessment area). For species breeding onsite, the ED is assumed to be 0.65 (i.e., receptors potentially spend up to 65% of the year on the assessment area). For migratory summer and winter visitors, the ED is assumed to be 0.25. The most conservative ED is chosen from the functional group members to represent the functional group ED.

Food intake rates (grams dry weight per day) for passerine birds, nonpasserine birds, rodents, herbivores, all other mammals, and insectivorous reptiles were estimated using the following allometric equations (Nagy 1987):

$$Food\ intake\ rate = 0.398\ BW^{0.850}\ (passerines) \quad (7\text{-}1)$$

$$Food\ intake\ rate = 1.110\ BW^{0.445}\ (desert\ birds) \quad (7\text{-}2)$$

$$Food\ intake\ rate = 0.648\ BW^{0.651}\ (all\ birds) \quad (7\text{-}3)$$

$$Food\ intake\ rate = 0.583\ BW^{0.585}\ (rodents) \quad (7\text{-}4)$$

$$Food\ intake\ rate = 0.577\ BW^{0.727}\ (mammalian\ herbivores) \quad (7\text{-}5)$$

$$Food\ intake\ rate = 0.235\ BW^{0.822}\ (all\ other\ mammals) \quad (7\text{-}6)$$

$$Food\ intake\ rate = 0.15\ BW^{0.874}\ (desert\ mammals) \quad (7\text{-}7)$$

$$Food\ intake\ rate = 0.013\ BW^{0.773}\ (reptile\ insectivores) \quad (7\text{-}8)$$

where

BW = body weight in grams.

Table 7-14. WAG 5 species parameters.

Functional groups	PP[a]	PV[b]	PS[c]	ED[d]	IR[e] (kg/day)	Nagy equation	BW[f] (kg)	HR[g] (Ha)	WI[h]
Avian herbivores (AV122)	0.00E+01	9.07E-01	9.30E-02	1.00E-00	1.46E-03	All birds	3.50E-03	5.18E-00	1.33E-03
Avian insectivores (AV210)	9.80E-01	0.00E+01	2.00E-02	6.50E-01	2.90E-03	All birds	1.00E-02	8.38E-00	2.70E-03
Avian insectivores (AV210A)	9.70E-01	0.00E+01	3.00E-02	6.50E-01	3.89E-03	Passerines	1.46E-02	2.39E-00	3.48E-03
Avian insectivores (AV222)	9.07E-01	0.00E+01	9.30E-02	1.00E-00	3.07E-03	All birds	1.09E-02	3.80E-01	2.86E-03
Avian carnivores (AV310)	9.80E-01	0.00E+01	2.00E-02	1.00E-00	1.61E-02	All birds	1.39E-01	2.18E+02	1.57E-02
Northern goshawk	9.80E-01	0.00E+01	2.00E-02	2.50E-01	6.00E-02	All birds	1.05E-00	2.13E+02	6.10E-02
Peregrine falcon	9.80E-01	0.00E+01	2.00E-02	2.50E-01	4.96E-02	All birds	7.82E-01	3.31E+01	5.00E-02
Avian carnivores (AV322)	9.80E-01	0.00E+01	2.00E-02	1.00E-00	7.44E-03	All birds	4.25E-02	9.00E-00	7.11E-03
Bald eagle	9.80E-01	0.00E+01	2.00E-02	2.50E-01	1.60E-01	All birds	4.74E-00	4.94E+02	1.67E-01
Ferruginous hawk	9.80E-01	0.00E+01	2.00E-02	6.50E-01	6.19E-02	All birds	1.10E-00	5.60E+02	6.29E-02
Loggerhead shrike	9.80E-01	0.00E+01	2.00E-02	6.50E-01	7.44E-03	All birds	4.25E-02	4.57E-00	7.11E-03
Avian carnivores (AV322A)	9.70E-01	0.00E+01	3.00E-02	2.50E-01	1.73E-02	All birds	1.55E-01	1.00E+01	1.69E-02
Burrowing owl	9.70E-01	0.00E+01	3.00E-02	2.50E-01	1.73E-02	All birds	1.55E-01	1.00E+01	1.69E-02
Avian omnivores (AV422)	6.27E-01	2.80E-01	9.30E-02	1.00E-00	1.13E-02	All birds	8.02E-02	1.10E+01	1.09E-02
Mammalian herbivores (M121)	0.00E+01	9.80E-01	2.00E-02	2.50E-01	3.14E-01	Mammal herbivore	5.80E-00	1.10E+01	4.82E-01
Mammalian herbivores (M122)	0.00E+01	9.37E-01	6.30E-02	1.00E-00	3.30E-03	Mammal herbivore	1.10E-02	2.30E-01	1.71E-03
Mammalian herbivores (M122A)	0.00E+01	9.23E-01	7.70E-02	1.00E-00	4.27E-03	Mammal herbivore	1.57E-02	3.00E-01	2.35E-03
Pygmy rabbit	0.00E+01	9.80E-01	2.00E-02	1.00E-00	4.53E-02	Mammal herbivore	4.04E-01	2.80E-01	4.38E-02
Mammalian insectivores (M210)	9.80E-01	0.00E+01	2.00E-02	2.50E-01	2.11E-03	Rodents	9.03E-03	2.39E-00	1.43E-03
Mammalian insectivores (M210A)	9.80E-01	0.00E+01	2.00E-02	1.00E-00	1.43E-03	Rodents	4.65E-03	2.39E-00	7.88E-04
Townsend's Western big-eared bat	9.90E-01	0.00E+01	1.00E-02	1.00E-00	2.37E-03	Rodents	1.10E-02	2.39E-00	1.71E-03
Small-footed myotis	9.90E-01	0.00E+01	1.00E-02	1.00E-00	1.44E-03	Rodents	4.69E-03	2.39E-00	7.94E-04
Long-eared myotis	9.90E-01	-1.00E-02	2.00E-02	1.00E-00	1.77E-03	Rodents	6.65E-03	2.39E-00	1.09E-03
Mammalian insectivores (M222)	9.76E-01	0.00E+01	2.40E-02	1.00E-00	1.66E-03	Rodents	6.00E-03	1.24E-01	9.91E-04
Merriam's shrew	9.76E-01	0.00E+01	2.40E-02	1.00E-00	1.66E-03	Rodents	6.00E-03	1.24E-01	9.91E-04
Mammalian carnivores (M322)	9.23E-01	0.00E+01	7.70E-02	1.00E-00	1.66E-02	All mammals	1.78E-01	1.30E+01	2.09E-02
Mammalian omnivores (M422)	8.04E-01	1.00E-01	9.40E-02	1.00E-00	3.06E-03	Rodents	1.70E-02	7.20E-01	2.53E-03
Reptilian insectivores (R222)	9.76E-01	0.00E+01	2.40E-02	1.00E-00	5.60E-05	Reptile insectivores	6.61E-03	1.17E-01	0.00E+01

Table 7-14. (continued).

Functional groups	PP[a]	PV[b]	PS[c]	ED[d]	IR[e] (kg/day)	Nagy equation	BW[f] (kg)	HR[g] (Ha)	WI[h]
Sagebrush lizard	9.76E-01	0.00E+01	2.40E-02	1.00E-00	5.60E-05	Reptile insectivores	6.61E-03	1.17E-01	0.00E+01
Reptilian carnivores (R322)	9.52E-01	0.00E+01	4.80E-02	1.00E-00	6.80E-03	Literature value[i]	1.50E-02	3.00E-00	0.00E+01
Plants	0.00E+01	0.00E+01	1.00E-00	1.00E-00					

a. PP = percentage of diet represented by prey ingested (unitless). Herbivores = 0% prey, total PV = PV-PS, carnivores = 0% vegetation, total PP = PP - PS); and omnivores =(1.00-PS-PV)/2 for representative species.

b. PV = percentage of diet represented by vegetation ingested (unitless).

c. PS = percentage of diet represented by soil ingested (unitless). Soil ingestion from Beyer, Connor, and Gerould (1994) and Arthur and Gates (1988) - (pronghorn, jackrabbit).

d. ED = exposure duration (fraction of year spent in the affected area) (unitless). Conventions: Residents - 0.05 to 1.00 (birds and migratory and transient mammals) 1.00 (small mammals); breeding – 0.05 to 0.65 (birds and migratory and transient mammals); summer visitors - 0.05 to 0.25; winter visitors - 0.05 to 0.25.

e. IR = ingestion rate [derived using allometric equations 7-1 through 7-9 based on body weight (Nagy 1987)] (kg/day).

f. BW = receptor-specific body weight (kg). Mammalian body weights primarily from Burt and Grossenheider (1976), the general literature and EPA Exposure Factors Handbook (December 1993) for some species. Avian body weights from Dunning (1993).

g. Home ranges from Hoover and Wills (1987) and the general literature. Unknown = defaulted to an SUF of 1.0 (i.e., assumes 100% site use).

h. WI = water ingestion rates derived using allometric equation (EPA December 1993).

i. Compiled from Diller and Johnson (1988).

An equation for ingestion rates for carnivorous reptiles (R322) was compiled from Diller and Johnson (1988).

$$Food\ intake\ rate = 0.01\ BW^{1.6}\ (carnivorous\ reptiles) \tag{7-9}$$

where

BW = body weight in kilograms.

Because different allometric equations may apply to different species within a group, the equations representative of all mammals and avians were used to calculate the ingestion rate (IR) for the functional groups. Exposure of each functional group was calculated using the best available estimates for species-specific exposure parameters. Each receptor was evaluated individually. Potential exposure for these species was determined based on the species' life history and feeding habits. Quantification of exposures used species-specific numerical exposure factors including body weight, ingestion rate, and the fraction of diet composed of vegetation or prey, and soil consumed from the affected area. Species parameters used to model intakes by the functional groups are presented in Table 7-14. These values are derived from the various key species in the functional groups. The parameters in Table 7-14 are the maximum percent prey, percent vegetation, percent soil, exposure duration, the ratio of minimum ingestion rate (IR) to body weight, and home ranges for each functional group because these values were the most conservative. Percent soil ingestion rate values are derived from the *Wildlife Exposure Factors Handbook* (EPA December 1993) and Beyer, Connor, and Gerould (1994) and site-specific data where available.

7.3.2.1 Exposure to Nonradiological Contaminants. The exposure equation used to calculate average daily intake is used to calculate the dose to functional group and T/E species. For example, dose intake in mg/kg body weight-day can be estimated using the following equation, as adapted from the EPA *Wildlife Exposure Factors Handbook* (EPA December 1993):

$$EE_{tot} = \frac{\left[(PP \times CP) + (PV \times CV) + (PS \times CS)\right] \times IR \times ED \times SUF}{BW} \tag{7-10}$$

where

E_{Etot} = estimated exposure from all complete exposure pathways (mg/kg body weight-day)

PP = percentage of diet represented by prey ingested (unitless)

CP = concentration of contaminant in prey item ingested (mg/kg)

PV = percentage of diet represented by vegetation ingested (unitless)

CV = concentration of contaminant in vegetation ingested (mg/kg)

PS = percentage of diet represented by soil ingested (unitless)

CS = concentration of contaminant in soil ingested (mg/kg)

IR = ingestion rate (kg/day), food intake rate (g/day) divided by 1,000 g/kg

ED = exposure duration (fraction of year spent in the affected area) (unitless)

BW = receptor-specific body weight (kg)

SUF = site usage factor (site area divided by home range; cannot exceed 1) (unitless).

The concentration of contaminant in prey can be estimated using the equation:

$$CP = CS \times BAF \quad (7\text{-}11)$$

where

CP = concentration in prey ingested (mg/kg)

CS = concentration of contaminant in soil (mg/kg)

BAF = contaminant-specific bioaccumulation factor (unitless).

The concentration of contaminant in vegetation (CV) can be estimated using the equation:

$$CV = CS \times PUF \quad (7\text{-}12)$$

where

CV = concentration in vegetation (mg/kg)

CS = concentration of contaminant in soil (mg/kg)

PUF = contaminant-specific plant uptake factor (unitless).

Contaminant-specific PUFs (Baes et al. 1984) and concentration factors (CFs) for nonradionuclide contaminants are presented in Table 7-15. Concentration factors for metals were developed as discussed in Appendix H. The log of PUF and CFs for organics is estimated using 1.588-0.578 log K_{ow}, and -7.735 + 1.033 log K_{ow}, respectively (Travis and Arms 1988). Log partitioning coefficients (K_{ow}) were taken from the *Groundwater Chemicals Desk Reference* (Montgomery and Welkom 1990).

7.3.2.2 Uncertainty Associated with Functional Groups. The selection of receptor parameters was designed to ensure that each member of the functional groups was conservatively represented. Because all members of a functional group are considered similar, it is reasonable to assume that all members of a group will be equally exposed to site-related contaminants. Quantification of dose for each functional group is expected to provide sufficient data to assess the general condition of the ecosystem and to be adequately protective of the majority of species potentially inhabiting WAG 5. In addition, sensitive species are included in the list of receptors for which dose is calculated. Hence, uncertainty associated with the selection of receptor parameters is expected to minimally influence dose estimates.

7.3.2.3 Uncertainty Associated with the Ingestion Rate Estimation. Using food intake rates in dry weight/day may result in an overestimate of intake rates because dry weights will contain more contamination/unit of vegetation. Intake (ingestion) estimates used for the terrestrial receptors are based on data in the scientific literature, when available. Food ingestion rates are calculated by use of the allometric equations reported in Nagy (1987). Uncertainties associated with the use of allometric

Table 7-15. Plant uptake factors and bioaccumulation factors for WAG 5 nonradionuclide contaminants (unitless).

	PUF[a]	BAF for Insectivores[b]	BAF for Carnivores[c]	BAF for Omnivores[d]
Inorganics[e]				
Antimony	2.0E-01	9.0E-01	5.5E-03	9.0E-01
Arsenic	4.0E-02	1.0E+00	4.0E-02	1.0E+00
Barium	1.5E-01	1.0E+00	5.5E-01	1.0E+00
Cadmium	5.5E-01	1.1E+00	1.9E+00	1.9E+00
Chromium	1.9E-01	6.0E-02	2.0E-01	2.0E-01
Cobalt	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Copper	4.0E-01	1.0E+00	2.0E-01	1.0E+00
Fluoride	1.0E+00	1.0E+00	1.0E+00	1.0E+00
Lead	4.5E-02	3.0E-01	6.0E-01	6.0E-01
Manganese	2.0E+00	1.0E+00	2.5E-01	1.0E+00
Mercury	9.0E-01	4.0E-01	7.0E-01	7.0E-01
Nickel	6.0E-02	1.0E+00	6.0E-03	1.0E+00
Selenium	2.5E-02	1.0E+00	2.5E-02	1.0E+00
Silver	4.0E-01	1.0E+00	4.0E-01	1.0E+00
Thallium	4.0E-02	1.0E+00	1.0E+00	1.0E+00
Vanadium	5.5E-03	1.0E+00	1.0E+00	1.0E+00
Zinc	1.5E+00	1.0E+00	7.0E-01	1.0E+00
Organics[f]				
Aroclor-1248	1.3E-02	4.0E-04	4.0E-04	4.0E-04
Aroclor-1254	1.3E-02	4.0E-04	4.0E-04	4.0E-04
Benzo(a)pyrene	1.2E-02	4.1E-04	4.1E-04	4.1E-04
Xylene	5.0E-01	2.2E-05	2.2E-05	2.2E-05

a. PUF = Plant uptake factor, appropriate for use with AV100 and M100 functional groups.

b. Bioaccumulation factors (BAFs) for insectivores, appropriate for AV200 and M200 functional groups.

c. BAFs for carnivores, appropriate for AV300 and M300 functional groups.

d. BAFs for omnivores, appropriate for AV400 and M400 functional groups.

e. Values and literature (see Appendix H) for inorganics are derived from Baes et al. (1984).

f. Values for organics come from allometric equations presented in Travis and Arms (1988).

NA = Not available

equations could result in either an overestimation or underestimation of ingestion rate resulting in either an overestimation or underestimation of the true dose rate.

7.3.2.4 Uncertainty Associated with the Receptor Site Usage. The calculation of dose incorporated the probability that the receptors may use or inhabit each site. The SUF is defined as the affected area (ha) divided by the home range (ha) of the receptor. If the home range of a given receptor is larger than the affected area, then it is reasonable to assume that the receptor may not spend 100% of its life within the site area. Incorporation of the SUF adjusts the dose to account for the estimated time that the receptor spends on the site. The less time spent on the site, the lower the dose. Home ranges for several functional groups are unknown and in these cases, the SUF equals 1. This may result in an overestimate of the potential exposure to these receptors.

7.3.2.5 Uncertainty Associated with the PUFs and BAFS. The advantages of using PUFs to estimate plant concentrations are that they are easy to use and require minimum data entry (i.e., the measured or estimated concentration of metal in soil and a PUF taken from the literature). A PUF of 0.01 indicates that the plant concentration should be 1/100th of the total concentration in soil. For the WAG 5 ERA, PUFs for metals are taken from Baes et al. (1984). Though preference is given to studies that reported the steady-state concentration of metals in plants at edible maturity, various soil properties are not considered and data for numerous plant species (both animal feeds and those consumed by humans) are combined. However, root uptake of metals is a complex process that depends on various soil properties (e.g., pH, the cation exchange capacity, and organic matter content) as well as the metal and type of plant involved. Therefore, the use of generic or crop-specific PUFs taken from the literature may not result in an accurate estimate of the concentration of metals in plants for all environmental conditions and species that may occur in WAG 5.

The PUFs for organics are estimated using the geometric mean regression equation (Travis and Arms 1988) and using log K_{ow} values. The reliability of estimated PUFs is directly related to the reliability of the K_{ow} values used for the organics. Because K_{ow} values can vary greatly, use of the regression equation (Travis and Arms 1988) to estimate a PUF for organics may result in either an overestimate or an underestimate of the true dose for organics.

A great deal of uncertainty is associated with the BAFs used to calculate dose. Very few BAFs are available in scientific literature because they must be both contaminant- and receptor-specific. The BAFs used for metals are discussed in Appendix H. The regression equation (Travis and Arms 1988) was used to calculate BAFs for the organic contaminants at WAG 5. An assumption that terrestrial receptors of concern accumulate metals and organics in a similar way and comparable degree to beef and dairy cattle was incorporated in the dose calculations. In the absence of specific BAFs, a value of 1 was assumed. This assumption could result in either an overestimate or an underestimate of the true dose from the contaminant, and the magnitude of error cannot be quantified. The terrestrial receptors of concern for WAG 5 may accumulate organics to a much larger or smaller degree than beef and dairy cattle; therefore, using the regression equation (Travis and Arms 1988) also could result in either an overestimate or an underestimate of the dose from the COPCs. In addition, the use of BAFs, as discussed in Appendix H, could result in either an overestimate or an underestimate of dose to ecological receptors at the site in the absence of site-specific data.

7.3.2.6 Uncertainty Associated with Soil Ingestion. The exposure assessment incorporates the percentage of soil ingested by each representative of the functional groups. Though food ingestion rates have the greatest effect on intake estimates, soil ingestion rates also could influence intake rates and, therefore, dose estimates. The EPA Wildlife Exposure Factors Handbook (EPA December 1993) and Beyer et al. (1994) were used to assign soil ingestion parameters to four of the 12 functional groups, and the percent of soil ingested was assigned to one species (Arthur and Gates 1988). Where information did

not exist in the literature on soil ingestion rates for terrestrial biota, soil ingestion rates were assumed to be 2% of the food ingestion rate for all burrowing mammals and birds that consume whole terrestrial prey and 1% for all other receptors. Estimating the percent soil ingested may result in either an overestimate or an underestimate of the dose because the effect of the estimated values on the overall dose outcome is dependent on the concentration of contaminant in the media of concern.

7.3.3 Ecological Effects Assessment

Ecological effects assessment consists of three elements:

- Selecting quantified critical exposure (QCE) levels
- Developing adjustment factors (AFs)
- Developing TRVs.

The following sections contain a general description of the procedures of ecological effects assessment and a discussions of each of the three elements.

7.3.3.1 General Procedures of the Ecological Effects Assessment. A TRV is defined as a dose for a receptor (including sensitive subgroups such as taxa under regulatory protection) that is likely to be without appreciable risk of deleterious effects from chronic exposure. Application of toxicity data derived from surrogate species introduces uncertainty into the risk assessment. The magnitude of this uncertainty depends largely on (1) the degree of taxonomic difference between the key and test species, (2) the conditions under which the toxicity data are obtained, and (3) the endpoint of interest (e.g., the chronic lowest-observed-adverse-effect level [LOAEL] or the no-observed-adverse-effect level [NOAEL]) and the endpoint measured (e.g., death). Adjustment factors are applied in the development of the TRVs in an attempt to offset the uncertainties associated with extrapolation of toxicity information from literature to site conditions.

The approach for TRV derivation used in the WAG 5 ERA was developed for use at the Rocky Mountain Arsenal Superfund site in Commerce City, Colorado (Ludwig et al. 1994) and is generally based on the EPA reference dose approach (Lewis, Lynch, and Nikiforov 1990). The approach is predicated on the development and application of AFs, which are extended to explicitly account for variations and uncertainties in data and necessary extrapolations from the data. The types of variation and extrapolation uncertainties explicitly quantified include the following:

- Variation in sensitivity among the members of a receptor population
- Uncertainty in extrapolating data from one taxon to another
- Uncertainty in using various effect levels to estimate no-effect-level receptors
- The inability of any single study to adequately address all possible adverse outcomes in a wild receptor population.

The approach developed for the Rocky Mountain Arsenal Superfund Site (Ludwig et al. 1994) offers several distinct advantages. By carefully identifying the specific types of adjustments needed in the extrapolation, the method permits maximum resolution of what each adjustment is intended to achieve. The method emphasizes consensual data-quality-based development of values for specific AFs rather than defaulting to arbitrary factors. It clearly discriminates between best estimates of the values of individual

factors and adjustment for overall uncertainty, including the uncertainty associated with the AFs themselves.

The TRV values used for arsenic, barium, beryllium, chromium, cobalt, copper, lead, manganese, mercury, silver, selenium, thallium, and vanadium for plants were taken directly from an Oak Ridge National Laboratory study on contaminants (Will and Suter 1995), and no AF values were assigned. The values presented in that paper are toxicological benchmarks for screening COPCs for effects on terrestrial plants in soil.

7.3.3.1.1 Selecting Quantified Critical Exposures—Toxicity reference value development is initiated by reviewing the available toxicological literature and relevant databases for each contaminant and functional group members to identify QCEs from the best available study. Studies considering nonlethal endpoints and reporting NOAELs are selected, if available. Studies that reflect reproductive competence are preferred because such endpoints are considered to best reflect the population-level impacts of greatest concern in the ERA. The following criteria are used to select QCEs:

- Experimental taxa should be as similar as possible to receptors at any applicable INEEL site, both physiologically and ecologically. For body size, feeding, and behavioral habits, anatomy, and physiology, the surrogate species should be matched as closely as possible to the receptors.

- The test exposure route and medium should be similar to that expected for receptors in the field. For most of the receptors at the INEEL, exposure media are limited to soil and dietary items (both animal and vegetable). Liquid intake is largely in the form of metabolic water. Dietary laboratory studies are, therefore, the most appropriate models for extrapolation. Gavage and drinking water studies will be considered if necessary, but they reduce confidence in the applicability of the studies.

- Long-term (preferably lifetime) exposures should be used because they are closest to exposure patterns occurring in the field.

- Experimental endpoints should represent ecologically significant effects at the population level. In general, the loss of a few individuals of a species is unlikely to significantly diminish the viability of the population or to disrupt the community or ecosystem of which the species is a part. Therefore, the fundamental unit for ERA is generally the population rather than the individual, with the exception of T/E species (EPA 1992). In general, the most appropriate endpoints for ERA are reproduction, neurological function, and growth and development. For species under regulatory protection, TRVs are based on the most sensitive nonlethal endpoints referencing specifically to individuals.

- Doses within the NOAEL-LOAEL bracket should be identified. If these data are not available, the following dose levels (in decreasing order of preference) may be used: chronic-nonlethal-adverse-effect-level > no-effect-level > frank-effect-level (including lethality). The definition of adversity requires considerable analysis of the potential ecological significance of the effects reported. For example, elevated liver weight or enzyme induction could represent an adaptive response rather than a toxic injury.

- Studies should be of high quality, which is defined as complete in design with adequate numbers of subjects and dose levels, lifetime duration, explicit analysis of experimental uncertainty, clear results, and well-justified conclusions.

If a single study cannot be selected (e.g., where only acute exposure, lethal endpoint studies are available), then an average of several studies of similar quality using the same or very similar species may be used. In averaging, extreme outliers (defined as greater than two standard deviations away from the mean) are excluded. Where similar endpoints are observed in more than one study of similar quality, the lowest QCE should be used.

For some chemicals, data in the toxicological literature are inadequate to enable selection of an appropriate QCE for certain receptors. Therefore, TRVs cannot be developed for these chemicals and receptors, and potential risks cannot be evaluated. Information was not located on the toxicological effects of all COPCs on reptilian receptors. Therefore, reptiles could not be evaluated for potential risk from exposure to contaminants in soil at WAG 5.

Information on the toxicological effects on avian receptors of the following COPCs was not located. Therefore, these contaminants could not be evaluated for potential effects to birds:

- Antimony
- Barium
- Benzo(a)pyrene
- Silver
- Xylene.

Information on the toxicological effects on plants of the following COPCs was not located. Therefore, these contaminants could not be evaluated for potential effects to vegetation:

- Benzo(a)pyrene
- Xylene.

7.3.3.1.2 Developing Adjustment Factors—The seven AFs for extrapolation from experimental studies to field exposures at the INEEL are defined as follows (Ludwig et al. 1994):

I = Intrataxon variability

R = Intertaxon variability

Q_1 = Certainty that the COPC actually causes the critical effect in the receptor and that it is an ecologically significant effect

Q_2 = Extrapolation from short- to long-term exposure durations

Q_3 = Extrapolation across endpoint types to estimate an NOAEL

U = Any residual uncertainty in the data evaluation process and estimation of other AFs based on data quality, study design, and known but otherwise unaccounted for extrapolation issues

M = Professional judgment to determine another uncertainty factor (M) that is < 10. The magnitude of the M depends upon the professional assessment of scientific uncertainties of the study and database not explicitly treated above; e.g., the completeness of the overall database of the number of animals tested. The default value of M is 1.

Values for these AFs are set based on the quality of the selected study in particular and of the database in general. Other potentially influential factors include the ecological circumstances of the

receptor, regulatory criteria and standards, background contaminant levels, and protection status. To prevent needless overestimation of risk, the maximal AF product (all AFs multiplied together) is scaled to the overall extrapolation error observed in experimental studies designed specifically to determine the uncertainty in such extrapolations. In one study (Barnthouse, Suter, and Rosen 1990), the range of maximal uncertainty necessary to permit extrapolation of various kinds of toxicity data for various taxa of fish at the population level was quantified. The types of toxicity data used included studies involving particular species of interest and other species for acute, partial life cycle, and full life-cycle exposures. The range of maximal uncertainty varied with the type of data used. The uncertainty ranged from approximately 200 to 400 (Barnthouse, Suter, and Rosen 1990). It is assumed that the degree of variability observed among fish taxa is similar to that occurring among other vertebrate taxa.

Based on a systematic review of all available information (Ludwig et al. 1994), a simple relative scale is developed consisting of "low," "medium," and "high" rankings for each AF, with adjustments made of the basis of specific inherent uncertainty or availability in the particular extrapolations. The quantitative valuation of this scale is designed to be constrained by an upper bound in the range of 200 to 400 and to use the most plausible values for each AF.

Specific values for these AFs and a brief description of criteria for their use are presented in Table 7-16. Values for all AFs except Q_1, and M are set at 1 (low), 2 (medium), and 3 (high), with lower values generally representing greater confidence that the QCEs correspond well with "safe" doses for receptors. The factor Q_1, which expresses the degree of certainty that the experimental effect will not occur in the field or is not of ecological significance, runs on a positive scale equivalent where 0.1 represents high certainty that the effect either does not occur in the receptor or is ecologically irrelevant. A value of 0.5 represents moderate certainty that the effect does not occur or is irrelevant, and a value of 1 represents reasonable certainty that the effect will occur in the receptor species and is ecologically significant. The factor M is used to adjust uncertainty based on professional judgment. For example, M can be set at 1 if the medium of exposure in the QCE study is similar to field exposure media at this site (i.e., primarily food and soil ingestion). However, because a number of toxicological studies for metals used soluble salts in drinking water as a means of exposure and both the contaminant species and exposure matrix tend to maximize metal absorption (Steele et al. 1990; Griffin and Turck 1991; Witmer, Harris, and Shupack 1991), M may be set at 0.5 to conservatively represent the significantly lower bioavailability of the metal species associated with soils and dietary items in the natural environment. Without M being greater than 1.0, the maximum product of the seven AFs is 243. This AF maximum represents the extent to which valid extrapolation of the data can be applied across experimental protocols or among taxa. More detailed information on the definition and valuation of these factors is available from the Rocky Mountain TRV study (Ludwig et al. 1994).

7.3.3.1.3 Developing Toxicity Reference Values—The third element in ecological effects assessment is the derivation of TRVs. Toxicity reference values were derived for each functional group by selecting the experimental study with the most appropriate QCE for a chemical and assigning numeric values for all AFs to account for uncertainties associated with extrapolation across species and exposure conditions.

The algorithm used for developing a TRV is:

$$TRV = \frac{QCE}{AF} \tag{7-13}$$

where

Table 7-16. Adjustment factor values and criteria for their use in developing toxicity reference values for the INEEL.

Adjustment Factor	Qualitative Ranking	Value	Criteria
I	Low	1	Variability is low.
	Medium	2	Variability is moderate or average.
	High	3	Variability is high, or information on variability is inadequate.
R	Low	1	The test organism and functional group, T/E species, and C2 species are in same taxonomic order and trophic category.
	Medium	2	The test organism and functional group, T/E species, and C2 species are in same trophic category but may be in different taxonomic order.
	High	3	The test organism and functional group, T/E species, and C2 species are in different trophic categories and taxonomic order.
Q_1	Low	0.1	The experimental endpoint is highly unlikely to occur in the field.
	Medium	0.5	The experimental endpoint is moderately unlikely to occur in the field.
	High	1	The experimental endpoint is likely to occur in the field.
Q_2	Low	1	The study was of chronic duration.
	Medium	2	The study was of subchronic duration.
	High	3	The study was of acute duration.
Q_3	Low	1	No observed adverse effect level (NOAEL).
	Medium	2	Lowest observed adverse effect level (LOAEL).
	High	3	The adverse-effect level or frank-effect level.
U	Low	1	Studies are of high quality.
	Medium	2	Studies are of reasonable quality.
	High	3	Studies have flawed design or incomplete information.
M	—	<10	Use professional judgment to determine another uncertainty factor (M).

QCE = quantified critical exposure level (unitless) QCE = quantified critical exposure level (unitless)

AF = [I] × [R] × [Q_1] × [Q_2] × [Q_3] × [U] × [M] (unitless).

Information used to derive TRVs for nonradioactive inorganic and organic contaminants is summarized in this section. A summary of TRVs for each contaminant, functional group, and sensitive species combination is presented in Appendix G for mammalian and avian receptors. The TRVs for

mammalian functional groups and nonsensitive species are summarized in Table G-1 in Appendix G. A summary of the TRVs for avian functional groups also is contained in Table G-2 in Appendix G. Shading in Tables G-1 and G-2 corresponds to the TRVs chosen for each functional group. Using the most appropriate study, when the test organisms and the receptor were in the same taxonomic order and trophic category (R = 1), the corresponding TRV was chosen, as shown in heavier shading. When the test organism and the functional group are in the same trophic category an R = 2 AF is used. Otherwise, the most appropriate TRV developed using R = 3 was used. Little information was found describing the effects of COPCs on reptilian, invertebrate, or terrestrial plant receptors. When available, that information is summarized in Sections 7.3.4 and 7.3.5.

7.3.4 Development of Toxicity Reference Values for Inorganic Contaminants of Potential Concern

This section contains summaries of the information used to determine the TRVs for the WAG 5 inorganic COPCs:

- Antimony
- Arsenic
- Barium
- Cadmium
- Chromium
- Cobalt
- Copper
- Fluoride
- Lead
- Manganese
- Mercury
- Nickel
- Selenium
- Silver
- Thallium
- Vanadium
- Zinc.

The development of TRVs for the studies identified for each COPC is contained in Appendix G.

Many of the inorganic contaminants are metals. Soils represent the most concentrated source of metals in the terrestrial environment. The health risks posed by trace metals in soils are not determined solely by their quantity. A number of contaminant, environmental, and biological conditions and processes influence the accessibility and availability of metals to organisms and, therefore, the toxicological significance of the metals. First, speciation is a major determinant of the fate, bioavailability, absorption, and toxicological characteristics of metal compounds. Second, the distribution coefficient between soil and water (K_d) depends on both the properties of the metal and the composition of the soil. This coefficient also governs the bioavailability of a metal to organisms contacting the soil, with weakly bound metals highly bioavailable and more strongly bound metals less bioavailable. Other influential factors include (1) the characteristics of the interface (e.g., with the lungs, skin, and the intestines), (2) the reactivity of the metal with the interface, and (3) the concurrent presence of other metals or other substances that may stimulate or inhibit metal uptake.

7.3.4.1 Antimony. Antimony is found in small amounts in the earth's crust (ATSDR 1992a). Antimony is a brittle metal that is not readily fabricated and has no significant use in its unalloyed state. It is alloyed with lead and other metals to increase their hardness, mechanical strength, corrosion resistance, and electrochemical stability or decrease their coefficient of friction. The most common end-use of antimony compounds is antimony trioxide for fire retardation. Antimony trioxide in a suitable organic solvent is used as a fire retardant for plastics, textiles, rubber, adhesives, pigments, and paper (U.S. Bureau of Mines 1989).

Antimony is released to the atmosphere in the form of particulate matter or adsorbed to particulate matter. It is dispersed by wind and removed by gravitational settling and dry and wet deposition (Schroeder et al. 1987).

Antimony released into waterways is generally associated with particulate matter; it is transported to and settles out in areas of active sedimentation such as where a river empties into a lake or bay (Beijer and Jernolov 1979).

Antimony may accumulate with heavy elements in carbonaceous shales or become sorbed on clays and hydrous oxides. Antimony may be volatilized when stibine (SbH_3) or its methylated derivatives are formed during the reduction of antimony in soils. Antimony has an affinity for clay and other mineral surfaces. Less than 10% of the antimony in sediments from both contaminated and uncontaminated sediments was found to oxidize organic matter easily (Callahan et al. 1979). Since antimony has an anionic character in aqueous solution, it probably has little affinity for complexation with humic and fulvic acids.

Antimony causes a number of toxic effects in animals, including suppression of weight gain, shortened life span, and damage to liver, heart, thyroid, and kidneys. Trivalent compounds (e.g., antimony trioxide, antimony trisulfide) are about 10 times more toxic than pentavalent forms. The gastrointestinal absorption of trivalent antimony is about 15 to 36% (Weitz and Ober 1965; van Bruwaene et al. 1982; Gerber, Mays, and Eykens 1982). The acute toxicity of antimony trioxide is low, with an oral LD_{50} in rats of greater than 20 g/kg (Smyth and Carpenter 1948).

In chronic studies, 5 mg/L potassium antimony tartrate (approximately 0.35 mg/kg-day) in drinking water is associated with slightly decreased life spans in rats (Schroeder, Mitchner, and Nasor 1970) and female mice (Schroeder et al. 1968; Kanisawa and Schroeder 1969). Endpoints examined in these chronic (lifetime) studies included growth and body weight, median life span, longevity, tumor incidence, and histopathology. Other ecologically relevant endpoints (e.g., reproduction) were not examined, and only one dose was administered. Though rats appeared to be more sensitive than mice in these studies, the effects reported are of questionable ecological significance.

7.3.4.2 Arsenic. Arsenic is a metalloid element that is widespread in all environmental media, making up about 0.0005% of the earth's crust. Arsenic is commonly present in living organisms and is constantly being oxidized, reduced, or metabolized. Many arsenic compounds are readily soluble in soil, making them available for plant uptake or for reduction by organisms or chemical interactions. Biological uptake of arsenic results in measurable quantities of reduced or methylated arsenic forms. Arsenic occurs naturally in all environmental media. Arsenic has four valence states: -3, 0, +3, and +5. Arsines and methylarsines, which are characteristic of compounds in the –3 state, are unstable in air. Most arsenicals degrade to yield arsenate, though arsenate may form under anaerobic conditions. Biotransformation of these compounds may occur and yield volatile arsenicals. The dominant form of arsenic present in aerobic soils is As+5, while As+3 is the primary species in anaerobic soils. Inorganic arsenic is more mobile than organic arsenicals and thus is more likely to leach into surface or groundwater. Trivalent species are generally more toxic, more soluble, and more mobile than pentavalent

forms. Soil microbes can metabolize arsenic to volatile arsine forms. The half-life of arsenic in soil is estimated to be 6.5 years for arsenic trioxide to 16 years for lead arsenate. Soils with high organic matter content, low pH, low phosphate, and low mineral content readily sorb arsenates. In air, most arsenic particulate contains inorganic arsenic compounds, particularly As+3 compounds (Eisler 1988a).

At relatively low levels, arsenic stimulates growth and development in several plant species (Eisler 1988a). The bioavailability of arsenic depends on several factors including pH, soil texture, fertility level, and plant species. Inorganic arsenate is readily taken up by plants via the phosphate carrier mechanism. Therefore, plants tend to have a poor ability to distinguish arsenate from phosphate. In general, arsenic is most available to plants grown in coarse soils having little colloidal material and a low ion-exchange capacity. Conversely, fine soils high in clay, organic matter, iron, calcium, and phosphate tend to retard the bioavailability of arsenic to plants (NRCC 1978). The accumulation of arsenic in plants tends to be directly correlated with the amount of arsenic in the dissolved fraction versus total arsenic concentrations (NRCC 1978).

The potential toxicity of arsenic to any organism is dependent on its chemical form. Inorganic arsenicals are generally more toxic than organic arsenicals, and trivalent forms are more toxic than pentavalent forms. Toxicity is related to aqueous solubility. The order of toxicity (from greatest to least) is arsines, inorganic arsenites, organic trivalent compounds, inorganic arsenates, organic pentavalent compounds, arsonium compounds, and elemental arsenic (Eisler 1988a).

Chemical properties contributing to the toxicity of arsenic include its ability to bind to protein sulfhydryl groups and to substitute for phosphorus in some biochemical reactions. These chemical properties also may be responsible for the apparent toxicity of arsenic in several mammalian species (Frost 1983; Uthus 1992). In fact, arsenical feed additives are used to promote growth in a number of agricultural species (Eisler 1988a). Recent studies have suggested that arsenic has a physiological role in the formation of various metabolites of methionine metabolism (Uthus 1992). The arsenic requirement for growing chicks and rats is approximately 25 mg/kg diet (Uthus 1992). Species differences in the pharmacokinetic disposition of arsenic have significant effects on their sensitivity to its toxic effects. In addition, animals exposed to sublethal levels of arsenic can develop tolerance to subsequent exposures (Eisler 1988a).

A subacute study using domestic sheep was documented (Eisler 1988a) in which an NOAEL endpoint using 2.3 mg/kg-day was reported. An LOAEL of 1.5 mg/kg-day was reported in a chronic study using sodium arsenate in rats (Byron et al. 1967). The data did not show a good dose-response curve in the low-dose range. This study was used in the development of TRVs for rats.

The National Academy of Sciences reported an LD_{50} of 39 mg/kg-day using sodium arsenite in mallards.

The recommended screening benchmark concentration for phytotoxicity in soil for arsenic of 10 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

7.3.4.3 Barium. Barium is distributed all over the earth and occurs most frequently as barite. Barium is used as a carrier for radium, deoxidizer for copper, lubricant for anode rotors in X-ray tubes, in plants, soap, paper, rubber, in the manufacture of ceramics and glass, and as a heat stabilizer for plastics. Barium metal in the free state does not occur in nature, but it is found in zinc or iron ores. Barium is emitted by industrial processes involved in the mining, refining, and production of barium and barium-based contaminants and as a result of the combustion of coal and oil.

In the atmosphere, barium is likely to be present in particulate form (EPA 1984). Although chemical reactions may cause changes in speciation of barium in air, the main mechanisms for the removal of barium compounds from the atmosphere are likely to be wet and dry deposition (EPA 1984).

Barium is not very mobile in most soil systems. Barium mobility in soil is reduced by the precipitation of barium carbonate and sulfate. Humic and fulvic acid have not been found to increase the mobility of barium (EPA 1984). Barium is taken up, retained, and excreted in mammals in much the same way as calcium compounds.

Little information regarding the toxicity of barium is available. Its acute toxicity is low, with LD_{50}s in experimental animals consistently greater than 100 mg/kg (ATSDR July 1992). High barium concentrations (2 to 10 ppm) in human drinking water have been reported to be associated with elevated cardiovascular mortality, hypertension, and other cardiovascular effects (ATSDR July 1992).

Results in animal studies indicate that acute, intermediate, and chronic oral exposure to barium is not associated with any adverse hematological effects. Developmental effects reported in a study by Tarasenko, Pronin, and Silaev (1977) in rats reported effects in offspring included increased mortality, increased leukocyte count, disturbances in liver function, and increased urinary excretion of hippuric acid.

Increased blood pressure, depressed cardiac contractility and conduction, and lower cardiac ATP content were observed in rats chronically exposed to barium concentrations of 10 to 100 mg/L in drinking water (Perry et al. 1983, 1985, 1989; Kopp et al. 1985). The NOAEL exposure level identified in these studies was 1 mg/L, or approximately 0.5 mg/kg/day.

No information on the toxicological effects of barium on avian receptors was located.

7.3.4.4 Cadmium. Cadmium is a silver-white, blue-tinged, lustrous metal that is insoluble in water, though its chloride and sulfate salts are relatively soluble in water. The availability of cadmium in soils depends on the soil pH, cation exchange capacity, chemical speciation, and many other factors. Adsorption and desorption processes tend to influence the concentration of cadmium in natural waters. Adsorption and desorption occur rapidly in soil. Cadmium tends to remain in the upper portion of the soil profile. Its bioavailability depends on adsorption/desorption rates, pH, and speciation. Cadmium uptake by plants is influenced by the concentration of calcium, sulfides, and sulfites present in the soil. Calcium and cadmium are considered to have the same uptake site; thus, levels of calcium present in soil could limit the amount of cadmium taken up by plants. Cadmium availability to plants is affected by the oxidation-reduction (redox) potential and pH. Humus-bound and sorbed cadmium contributes to the plant available pool. Availability may be reduced by higher organic matter content and higher cation exchange capacity (Eisler 1985a).

Cadmium is found naturally in the environment from chemical weathering of rocks. It is generally found in soil as the free cadmium compounds (ATSDR 1993a). There is no evidence that cadmium is biologically essential (Eisler 1985a). Cadmium is not reduced or methylated by microorganisms (ATSDR 1993a). Birds and mammals are comparatively resistant to cadmium toxicity as compared to aquatic species. Sublethal effects of cadmium include growth retardation, anemia, and testicular damage (Hammons et al. 1978 [as cited in Eisler 1985a]). Cadmium readily reacts with sulfhydral groups and may inhibit enzymatic reactions (Eisler 1985a). Bioaccumulation of cadmium has been reported in aquatic systems; however, only lower trophic levels are reported to exhibit biomagnification (Eisler 1985a). Accumulation of cadmium in avian species has been reported in liver and kidneys.

Toxicity reference values were developed using a multigeneration rat reproduction study by Wills, Groblewski, and Coulstone (1981) in which an LOAEL of 5 mg/kg-day was established.

Chickens exposed to cadmium in the diet had reduced growth rates in a study by Pritzl et al. (1974). This study was used to derive a TRV for avian receptors. Behavioral changes were observed in young American black ducks when parents were fed 4 ppm cadmium for 4 months before egg laying [Heinz and Haseltine 1983 (as cited in Eisler 1985a)].

For invertebrates, a study on the toxicity of cadmium nitrate to the isopod (*Porcellio scaber*) was used to develop a TRV. The study reports a critical concentration of 100 μg/g cadmium in food on a dry weight basis for reproduction (Hopkin and Hames 1994).

The recommended screening level toxicological benchmark of 3 mg/kg for the phytotoxicity in soil for cadmium was used as the TRV (Will and Suter 1995).

No information on the toxicological effects of cadmium on reptilian receptors was located.

7.3.4.5 Chromium. Chromium is a multivalent element and can exist in the +2, +3, and +6 oxidation states. The latter two, chromium(III) and chromium(VI), are the most stable in the environment. In soils and sediments, chromium is influenced by oxidation and reduction reactions and can be adsorbed on the mineral and organic exchange complex or exist as a coating in iron and manganese hydrous oxide particles. Moreover, chromium may remain in solution in the pore water phase, or may become chelated by an organic liquid or precipitated (Adriano 1986; Callahan et al. 1979). The sorption of chromium(VI) by hydrous metals oxides and other soil mineral components decreases as pH levels increase. The presence of other anions (e.g., sulfate and phosphate) significantly affects the extent of adsorption by competing for adsorption sites. The formation of ion pairs, such as dissolved calcium chromate, also may reduce the extent of adsorption. In contrast to chromium(VI), the sorption of chromium(III) increases as pH units increase. In general, it appears from laboratory studies that chromium(III) is adsorbed more heavily than chromium(VI). Organic material also may be an important adsorbent in sediments and soils. Slight enrichment of chromium occurs in the humic fraction. Typically, in normal, well-drained soils, the great majority of chromium is in the form of chromium(III).

Chromium(VI) is generally more toxic than chromium(III). Though most chromium(VI) is reduced to chromium(III) in the acidic environment of the stomach (Donaldson and Barreras 1966), chromium(VI) compounds are absorbed significantly more efficiently from the gastrointestinal tract (2 to 10% of administered dose) than chromium(III) compounds (Outridge and Scheuhammer 1993). Once absorbed, chromium(VI) is quickly reduced to the trivalent form. The damaging effects of chromium(VI) are caused by its greater membrane permeability, which allows it to cross biological membranes and oxidize cellular components not normally accessible to chromium(VI). Therefore, the differences in systemic toxicity are primarily attributable to differential solubilities and absorption rates of the two valence states (Franchini and Mutti 1988).

The mobility of chromium(VI) and the limited supply of extracellular reductants cause chromium(VI) to be distributed more widely in the body than chromium(III). The intracellular reduction of chromium(VI) to chromium(III) generates unstable intermediate chromium(V) and chromium(IV) ions, active oxygen species (hydroxyl and superoxide radicals, single oxygen), and thiyl and organic radicals that are responsible for the cytotoxicity, mutagenicity, and carcinogenicity of the hexavalent form (reviewed by Manzo et al. 1992; Cohen et al. 1993; O'Flaherty 1993; Outridge and Scheuhammer 1993).

Chromium exhibits a pattern of biominification rather than biomagnification in ecological food webs. Because the speciation of chromium(VI) taken up by plants is poorly understood, it is assumed to be the primary form of exposure to herbivores. However, chromium(VI) is immediately converted to chromium(III) in animal tissues. Therefore, carnivorous receptors will be primarily exposed to the less toxic trivalent form. The development of TRVs based on chromium(VI) for receptors higher in the food

chain is thus highly conservative, and will tend to result in an overestimate of chromium-related risk to these receptors.

In a study of chromium toxicity (Rosomer et al. 1961), a subchronic NOAEL of 100 mg/kg in the diet for chickens was reported. This information is used to estimate the TRV for avian functional groups.

Pregnant female mice receiving 250 mg/L potassium dichromate in drinking water throughout gestation showed no clinical signs of toxicity, but produced significantly fewer viable offspring (Trivedi et al. 1989). In dogs, 6 mg/L in drinking water (approximately 0.3 mg/kg/day) was a chronic NOAEL (Steven et al. 1976 [cited in Eisler 1986]). A similar level was without observable effects in a study of chronic toxicity (Anwar et al. 1961). Based on these results, TRVs were derived for mammalian functional groups.

Chromium was assessed as chromium(III) since chromium is not expected to persist in the environment at the INEEL in the chromium(VI) form (Bartlett and Kimble 1976; Rai, Eary, and Zachara 1989). Sample data collected from 10 grid locations at PBF-10 (a dried pond site) for both chromium(VI) and (III) support this assumption. The average ratio of the chromium(VI) to (III) soil concentrations is 0.0085 (ranging from 0.00017 to 0.053). The ratio of the minimum chromium(VI) EBSL (0.162 mg/kg) to the minimum chromium(III) EBSL (3.25 mg/kg) is 0.05. Therefore, it is unlikely that chromium(VI) would pose a risk unless chromium(III) first was shown to be a risk. For example, if a total chromium site concentration of 32.5 mg/kg was detected, when compared to the chromium(III) minimum EBSL, the screening hazard quotient would equal 10 (32.5 mg/kg divided by 3.25 mg/kg). However, based on the average ratio of chromium(VI) to chromium(III) (as calculated from PBF-10 data) the soil concentration of chromium(VI) would be 0.28 mg/kg (i.e., 0.0085 times 32.5 mg/kg). The screening hazard quotient for chromium(VI) would equal 1.73—that is, the chromium(VI) soil concentration of 0.28 mg/kg divided by the minimum EBSL of 0.162 mg/kg.

7.3.4.6 Cobalt. Cobalt is a dietary essential for ruminants and horses in which it is incorporated into vitamin B-12. Signs of cobalt deficiency in cattle and sheep are loss of appetite, body weight loss, emaciation, and anemia. Cobalt deficiency is more likely than cobalt toxicosis.

Environmental exposures to high levels of cobalt rarely occur. Characteristic signs of chronic toxicosis for most species are reduced feed intake and body weight, emaciation, anemia, hyperchromemia, debility, and increased liver cobalt (Turk and Kratzer 1960). A study by Brewer (1940) in which cobalt was mixed with the food of dogs in amounts equivalent to 5, 10, 15, 20, and 30 mg at no time during the course of the 4-week study showed any toxic signs. Adding cobalt in the form of cobalt chloride to the diet at levels up to 200 ppm did not result in toxicosis in pigs fed a diet adequate in iron (Huck and Clawson 1976). In another study, Hill (1979) observed growth retardation and decreased resistance to infection in chicks fed cobalt in protein mixtures.

7.3.4.7 Copper. Copper is one of the least mobile of the trace elements and tends to be uniformly distributed in the soil horizon. Soil parameters that influence copper availability include pH, the cation exchange capacity, and organic matter content. Persistence of copper in soils is caused by binding to organic matter, the formation of oxides with iron and manganese, the presence of clay minerals, and soil pH. A pH of 6 or less increases the mobility and availability of copper in soil. Copper is one of the trace elements most extensively complexed by humic materials. Most copper is readily available to plants when the soil pH is below 6, especially in soils with low organic matter and humic material content. Sulfides, which may prevail in soils under reducing conditions, effectively precipitate copper and thereby reduce the bioavailable amount of copper. Biogenic ligands bind with copper, which results in the precipitation and sorption of copper. Copper is one of seven essential plant micronutrients. Copper in soil tends to strongly bind with organic matter, which limits its availability for uptake by plants.

Copper is widely distributed in nature and is an essential element for (1) the normal function of several critical enzymes and (2) the use of iron. Copper deficiency is, therefore, usually a greater health concern than copper excess. Copper absorption in the gastrointestinal tract is normally regulated by body stores. Absorbed copper is transported to the liver, where it may be incorporated into ceruloplasmin (a copper transport and donor molecule) and then is excreted into the plasma and stored as metallothionein or in lysosomes, or is excreted via bile (reviewed by Nederbragt, Van den Ingh, and Wensvoort 1984).

Depressed food intake, body-weight gain, egg number and weight, and organ weights are associated with copper excess in poultry (Stevenson and Jackson 1981). The pair-feeding study was conducted to determine whether these effects were associated with direct toxicity or the accompanying marked reduction in food intake (Stevenson and Jackson 1981). Body weight, food intake, organ weights, egg production, egg weight, clinical chemistry parameters, and organ copper, iron, and zinc concentrations were monitored in laying hens fed varying concentrations of copper in their diet for 6 weeks (Stevenson and Jackson 1981). An NOAEL of 24 mg/kg/day was identified and used to develop TRVs for avian functional groups.

High doses of copper have caused liver and kidney damage and anemia in a number of species. The stomach also is a target in rats and mice (Hebert et al. 1993). This well-designed subchronic feeding study examined histopathology, clinical pathology, reproductive toxicity, and tissue metal accumulation in males and females of both species. A QCE of 66 mg/kg/day (an NOAEL) was identified from this study and used to develop mammalian TRVs. A chronic study of young calves (Cunningham 1946) confirmed that they are susceptible to chronic doses of copper. The QCE from this study is 1.1 mg/kg-day.

A mammalian TRV also was derived from a chronic feeding study in mink (Aulerich et al. 1982). The purpose of this study was to determine whether copper supplements would improve growth and survival. Endpoints examined included the effects on growth, blood chemistry, reproductive performance, and kit survival and development. The QCE from this study is an NOAEL of 12.9 mg/kg/day.

The recommended screening benchmark concentration for phytotoxicity in soil for copper of 100 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

7.3.4.8 Fluoride. In addition to occurring naturally, fluorides are used as insecticides; in the manufacture of aluminum, steel, glass, cement, bricks, high octane gasoline, and phosphate fertilizers; and for water treatment (McKee and Wolf 1963). Fluoride compounds are relatively persistent in the environment and are not biodegradable.

Inorganic fluorides are generally highly irritating and toxic. Acute effects resulting from exposure to fluorine compounds are from hydrofluoric acid. Chronic fluorine poisoning, or fluorosis, occurs with cryolite, and consists of a sclerosis of the bones caused by a calcium fixation by fluorine. Ligament calcification also may occur. The teeth become mottled, and osteosclerosis and osteomalacia occur. Large doses can cause very severe nausea, vomiting, diarrhea, abdominal burning, and cramp-like pains. Fluoride is not taken up by the thyroid and does not interfere with iodine uptake. It can cause or aggravate attacks of asthma and severe bone changes, making normal movements painful. Some signs of pulmonary fibrosis have been noted (Sax and Lewis 1987).

The reproductive effects of fluoride, administered orally to mink, were studied (Aulerich et al. 1987). Five dose levels were administered. Fluoride at levels of up to 229 ppm had no adverse effects on reproduction. The survival of kits in the 385 ppm group was significantly reduced. These doses had been considered to be NOAELs and LOAELS, respectively. Because the study considered

exposure over 382 days including critical life stages (reproduction), these doses were considered to be chronic. An NOAEL of 31.37 mg/kg/day was established.

Researchers studied the effects of fluoride when orally administered to a screech owl for 5 to 6 months (Pattee, Wiemeyer, and Swineford 1988). Fertility and hatching success was reduced significantly by 232 ppm fluoride in the diet, and 56.5 ppm fluoride in the diet had no adverse effect. Because the study considered exposure during reproduction, these doses were considered chronic. An NOAEL of 7.8 mg/kg/day was established and used to develop a TRV.

No information on the toxicological effects of fluoride on reptile and invertebrate receptors was located.

7.3.4.9 Lead. Lead is a ubiquitous trace constituent in rocks, soils, plants, water, and air, with an average concentration of 16 mg/kg in the earth's crust (Eisler 1988b). Lead has four stable isotopes: Pb-204 (1.5%), Pb-206 (23.6%), Pb-207 (22.6%), and Pb-208 (52.3%). Lead occurs in four valence states: elemental (Pb), monovalent (Pb^{+}), divalent (Pb^{+2}), and tetravalent (Pb^{+4}). In nature, lead occurs mainly as Pb^{+2} and is oxidized to Pb^{+4}. Metallic lead is relatively insoluble in hard waters. Some lead salts are somewhat soluble in water. Of the organoleads, tetraethyllead and tetramethyllead are the most stable and are highly soluble in many organic solvents but are fairly insoluble in water. Both undergo photochemical degradation in the atmosphere to elemental lead and free organic radicals. Organolead compounds are primarily anthropogenic (Eisler 1988b).

Lead is neither essential nor beneficial to living organisms. Lead affects the kidneys, blood, bone, and the central nervous system. The effects of lead on the nervous system are both functional and structural. Lead toxicity varies widely with the form and dose of administered lead. In general, organolead compounds are more toxic than inorganic lead. In nature, lead occurs mainly as divalent, Pb^{2+}. Ingestion of lead shot by regulatory waterfowl is a significant cause of mortality among these species.

Hatchlings of chickens, quail, and pheasants are relatively tolerant to moderate lead exposure (Eisler 1988b). Dietary levels of 500 mg/kg had no effect on hatchling growth of these species, and levels at 2,000 mg/kg lead had no effect on survival (Hoffman et al. 1985 as cited in Eisler 1988b). For avian herbivores, a TRV was estimated using a study of mallards (Dieter and Finley 1978). Altricial species are generally more sensitive to lead than precocial species (Eisler 1988b) of avian insectivores. An oral study using European starlings (Osborn, Eney, and Bull 1983) was used to generate a TRV for trimethyllead chloride. Because organic lead compounds are generally more toxic than inorganic lead, the toxicity quotients generated using this TRV should be interpreted with caution. American kestrels (*Falco sparverius*) exposed to 50 mg/kg/day metallic lead in diets did not exhibit effects on survival or reproductive success (Colle et al. 1980). Using these studies, TRVs were developed for avian functional groups.

Studies of rats administered lead in drinking water (Kimmel et al. 1980), lead toxicity in calves (Zmudzki et al. 1983), and lead toxicity of dogs (DeMayo et al. 1982) were used to develop TRVs for mammalian receptors. A critical concentration of 2,000 g/g lead in food on a dry weight basis for reproduction was reported in a study on the toxicity of lead nitrate to the isopod (*Porcellio scaber*) (Hopkin and Hames 1994).

The recommended screening benchmark concentration for phytotoxicity in soil for lead of 50 mg/kg was used as the TRV for terrestrial plants (Suter, Will, and Evans 1993).

7.3.4.10 Manganese. The bioavailability of different forms of manganese varies considerably depending on different exposure conditions (ATSDR 1992b). There is potentially higher bioavailability of manganese from drinking water than food. Furthermore, various dietary factors as well as the form of manganese can have a significant bearing on the dose absorbed from the gastrointestinal tract. For instance, many constituents of a vegetarian diet (e.g., tannins, oxalates, phytates, fiber, calcium, and phosphorus) have been found to inhibit manganese absorption presumably by forming insoluble complexes in the gut. Thus, herbivores are more likely to be resistant to manganese toxicity. Also, the form of manganese can significantly influence toxicity. For example, mice receiving the two soluble forms of manganese (chloride and acetate salts) were found to gain significantly less weight than controls, while mice consuming the insoluble forms of manganese (carbonate and dioxide salts) appeared to actually gain slightly more weight than controls.

DiPaolo (1964) subcutaneously or intraperitoneally injected mice with 0.1 mL of an aqueous of solution 1% manganese chloride twice weekly for 6 months. A larger percentage of the mice exposed subcutaneously (24/36; 67%) and intraperitoneally (16/39; 41%) to manganese developed lymphosarcomas compared with controls injected with water (16/66; 24%). In addition, tumors appeared earlier in the exposed groups than in the control groups. The incidence of tumors other than lymphosarcomas (i.e., mammary adenocarcinomas, leukemias, injection site tumors) did not differ significantly between the exposed groups and controls.

A study reporting the minimum manganese requirements in chickens was used to derive a TRV of 2.9 mg/kg/day. Guinea fowl were found to have reduced hatchability and increased deformed embryos when fed diets deficient in manganese (Offiong and Abed 1980).

A dietary reproduction study in rats exposed to 250 ppm manganese (13 mg/kg/day) was used to develop a TRV of 1.1 mg/kg/day (Laskey, Rehnberg, and Hein 1982).

7.3.4.11 Mercury. Mercury exists in the environment in three oxidation states: the elemental state, +1 (mercurous) state, and +2 (mercuric) state. The factors that affect which species dominates in an environment are the redox potential and the pH of the system. Particle-bound mercury can be converted to insoluble mercury sulfide, which can be bioconverted into more soluble or volatile forms that may reenter the atmosphere or be taken up by biota and bioaccumulated in the terrestrial food chain. Mercury forms many stable organic complexes that generally are more soluble in organic matter than in water. Inorganic and organic particles strongly sorb mercury. Mercury can be transformed in the environment by biotic and abiotic oxidation and reduction, bioconversion of organic and inorganic forms, and photolysis. Mercury can be strongly concentrated by living organisms (Callahan et al. 1979). The chemistry of mercury in the environment is complex, not only because of its various oxidation states but also because of biotic and abiotic methylation and demethylation processes, complexation with organic and inorganic ligands, and the differential solubility and volatility of various forms. Because speciation is a major determinant of the fate, bioavailability, absorption, and toxicological characteristics of mercury compounds, lack of knowledge of the state of the mercury in INEEL soils is a large source of uncertainty in both exposure assessment and TRV development.

Though the generally more toxic organic forms of mercury are unlikely to persist in the environment, they (in particular, methylmercury) may be formed in biotic tissues and are known to biomagnify through ecosystems, particularly aquatic systems (reviewed by Wren 1986; Scheuhammer 1987). Thus, to ensure that mercury TRVs for the WAG ERA are protective of receptors at all levels of ecological organization, TRVs are developed from studies investigating the toxic effects of organic mercury. It is noted that this measure is highly conservative and will tend to result in an overestimate of risks for receptors lower in the food web because the majority of mercury in soil and plants (i.e., the majority of exposure to plants and soil-dwelling and herbivorous animals) is expected to be inorganic.

Because of its chemical stability and lipophilicity, methylmercury readily penetrates the blood-brain barrier. The central nervous system is thus a major target organ in both mammals and birds. However, reproductive effects have been reported at even lower doses. Methylmercury can be converted to inorganic mercury both in tissues and by microflora in the gut. The homolytic cleavage of the mercury-carbon bond leads to generation of reactive intermediates (e.g., methyl and metal radicals, which cause cellular damage) (reviewed by Wren 1986; Scheuhammer 1987; Manzo et al. 1992).

The effects of mercury on avian herbivores, insectivores, and carnivores were evaluated. For herbivores, the effects of organic mercury compounds on galliformes (e.g., domestic chickens, quail, and pheasants) have been investigated by several groups. However, no study was reviewed that identified an NOAEL. The lowest LOAEL for relevant endpoints (reproductive success) of several similar studies was found in a study of the effects of mercury to birds (Fimreite 1979). Reduced egg production, shell thickness, and hatchability in pheasants fed seed treated with organomercurial fungicide were observed. This study was selected over others because of its use of a wild species and lower dose levels. A TRV was derived from this study.

Three goshawks were fed a diet of chickens that had eaten wheat dressed with an organomercurial fungicide (Borg et al. 1970). Their tissues contained 10 to 40 ppm of mercury, mostly as methylmercury. The hawks died after 30 to 47 days, and their total mercury intake was about 20 mg/bird.

Two studies examined the effects of subchronic methylmercury exposure on the reproductive competence of male and female rats (Khera and Tabacova 1973; Khera 1973). The NOAEL identified for both sexes was 0.25 mg/kg/day. Much less information is available about methylmercury toxicity to herbivores. In a study of acute methylmercury toxicity in mule deer (*Odocoileus hemionus hemionus*) 17.88 mg/kg was said to be the lethal dose of 50% of organisms exposed (LD_{50}) (Eisler 1987a). A number of studies have examined the effects of chronic methylmercury ingestion on carnivorous mammals, particularly cats (e.g., Albanus et al. 1972; Charbonneau et al. 1976; Eaton, Secord, and Hewitt 1980) and mink (e.g., Aulerich, Ringer, and Iwamoto 1974; Wobeser, Neilson, and Schiefer 1976; Wren et al. 1987). The study of the chronic toxicity of cats was considered superior to other available studies because of its long duration (2 years), use of relatively large group sizes, detailed examination of endpoints, identification of both no-effect and effect levels, and administration of mercury via both contaminated fish and addition to diet (Charbonneau et al. 1976).

A TRV of 0.3 mg/kg was assigned for mercury for terrestrial plants based on the toxicological benchmark (Suter, Wills, and Evans 1993).

7.3.4.12 Nickel. Nickel is a naturally occurring silvery metal that is found in the earth's crust (ATSDR 1988). Organic complexing agents may restrict soil movement and the availability of nickel through the formation of organic-nickel complexes (ATSDR 1988). However, nickel ferrite appears to be the most probable nickel species to precipitate in soil. Nickel is continuously transferred between air, water, and soil via various natural processes including weathering, runoff, erosion, and leaching. Nickel is very persistent in both soil and water. In the atmosphere, nickel exists primarily in the aerosol form. Nickel can exist in water in various soluble and insoluble forms depending on the physicochemical properties of the water. The mobility of nickel in aqueous media is affected by complexation, precipitation and dissolution, oxidation and reduction, and adsorption and desorption. The average residence time of nickel in soil is estimated to be 2,400 to 3,500 years. Though nickel is very persistent in soil, it can leach into groundwater. The sorption of nickel to soils correlates with pH, total iron, and organic matter content. Organic complexing agents in soil tend to restrict the movement of nickel by forming organs-nickel compounds. Nickel is fairly mobile in soils with a low pH and cation exchange capacity (ATSDR 1988).

Small amounts of nickel can be essential for normal growth and reproduction (ATSDR 1988). Oral exposure to high concentrations of nickel has been reported to adversely affect the hematological system and reproduction.

Toxicity studies of chicks (Weber and Reid 1968) and of mallards (Eastin and O'Shea 1981) were used to develop TRVs for avian herbivores.

Rats fed 5 mg/kg/day nickel sulfate in a 2-year dietary study did not produce hepatic changes or altered body weights (Ambrose et al. 1976). This NOAEL was supported by a rat subchronic drinking water study conducted by American Biogenics Corp. (ABC 1986) and a rat reproductive study by Research Triangle Institute (RTI 1987). Using the 2-year study (Ambrose et al. 1976), a TRV for small mammals was developed. For mammalian herbivores, a subchronic study of cows that did not exhibit reduced food intake or growth rate when fed 250 mg/kg/day nickel carbonate (NAS 1980) was used to develop a TRV. A dietary study exposing dogs to 1,000 ppm nickel did not result in adverse effects (Ambrose et al. 1976) and was used to develop a TRV for mammalian carnivores.

No information on the toxicological effects of nickel on reptile or invertebrate receptors was located.

The recommended screening benchmark concentration for phytotoxicity in soil for nickel of 30 mg/kg was used as the TRV for terrestrial plants (Suter et al. 1993).

7.3.4.13 Selenium. Selenium is a critical nutrient and a key component of several enzymes (Eisler 1985b). It is often found in high concentrations in areas in which soils have been derived from Cretaceous rocks (Eisler 1985b). Selenium accumulates to high concentrations in certain species of plants (e.g., *Aster* spp. and *Astragalus* spp.) (Eisler 1985b). Livestock species ingesting these plants have been reported to exhibit toxic symptoms such as abnormal movements, labored breathing, dilated pupils, bloating, diarrhea, and rapid pulse. No effective treatment is known for counteracting the toxic effects of high levels of ingested selenium. Prolonged exposure to more moderate levels of selenium results in skin lesions involving alopecia, hoof necrosis and loss, emaciation and increased serum transaminases, and alkaline phosphatase in animals (TOXNET 1994). Selenium has been reported to cause growth retardation, decreased fertility, embryotoxicity, fetotoxicity, and teratogenic effects in animals (TOXNET 1994). Birds appear to be particularly susceptible to selenium, particularly in the area of reproductive success. Malformations in chickens and waterfowl have been widely reported (EPA 1993).

Selenium deficiency is often a greater threat to health than selenium poisoning (Eisler 1985b). Selenium deficiency has been documented in a variety of species including fish, quail, ducks, poultry, rats, dogs, domestic grazing animals, antelope, monkeys, and humans (Eisler 1985b). Selenium also can reduce the toxicity of other heavy metals such as thallium, arsenic, and copper (Wilber 1980).

The effects of sodium selenite in chickens, mallards, and black-crowned night herons were evaluated in studies by Ort and Latshaw (1978), Heinz et al. (1987), and Smith et al. (1988) to derive TRVs for avian receptors.

In a study by Rosenfeld and Beath (1954), selenium administered as potassium selenate to sires and pregnant rats through five breeding cycles did not affect reproduction, the number of young reared, or the reproduction of two successive generations of dams and sires in groups receiving 1.5 ppm selenium. Because no effect on growth in rats has been reported at concentrations of 1.6 to 4.8 ppm selenium in the diet (Halverson, Palmer, and Guss 1966), a reproductive endpoint was selected to develop a TRV. Selenium doses of as low as 3.2 mg/kg body weight have resulted in death in sheep (Eisler 1985b). A TRV was developed for mammalian herbivores using these data.

No information on the toxicity of selenium in reptile or invertebrate receptors was located.

The recommended screening benchmark concentration for phytotoxicity in soil for selenium of 1 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

7.3.4.14 Silver. The precious metal silver is relatively rare in the earth's crust and does not occur regularly in animal tissues. As a result, the toxicity of silver has been studied lightly. Approximately 1 to 10% of ingested silver is absorbed—as much as 18% may be retained. Silver-protein complexes accumulate in the liver, and biliary excretion (complexed with glutathione) is the major route of elimination. In most tissues, silver is deposited as large granules. With rare exceptions, these deposits are not associated with adverse effects. The LD_{50} of silver in rats is relatively high at 24 mg/kg (reviewed by Rungby 1990).

Silver causes a conditioned deficiency of selenium in rats, decreasing tissue levels of selenium, and the selenoprotein glutathione peroxidase (Ganther 1980). Silver ions complex strongly to sulfhydryl groups and cause preoccupation of hepatocellular membrane lipids (Rungby 1987; Shinogi and Maeizumi 1993). Because of its affinity for sulfhydryls, the degree of binding to cellular macromolecules and toxicity of silver is mitigated by induction of the divalent metal-binding protein metallothionein (Shinogi and Maeizumi 1993). Exposure of fetal and adult rats to silver results in deposition in the central nervous system (Rungby and Danscher 1983a, 1983b). Pyramidal cells in the developing hippocampus appears to be a sensitive target, which exhibits reduced cellular volume in both pre- and postnatally exposed rats (Rungby 1987; Rungby 1990).

The mammalian TRVs for silver are based on a subchronic study by Rungby and Danscher (1984) in which mice exposed to approximately 18 mg/kg/day were observed to be "hypoactive." Though silver deposits occurred in certain motor centers of the brain, no association between the concentration of deposits and the extent of hypoactivity was found.

No information on the toxicological effects of silver on avian, reptilian, amphibian, or plant receptors was located. The recommended screening benchmark concentration for phytotoxicity in soil for silver of 2 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

7.3.4.15 Thallium. Thallium is a nonvolatile heavy metal element that is not used extensively by industry and is mainly introduced into the environment as a waste product of other metals. Thallium can exist in the atmosphere as an oxide, a hydrazide, a sulfate, or a sulfide. Thallium is present in mono- or trivalent forms in the environment. Thallium(III) forms some organometallic compounds, and thallium(I) forms relatively few complexes with the exception of those with halogen, oxygen, and sulfur ligands. Thallium can be removed from solution by adsorption onto clay minerals, bioaccumulation, or (in reducing environments) precipitation of the sulfide. Increased pH values have been found to produce extensive thallium-humic acid interactions while lowering thallium-inorganic interactions. Thallium may be bioconcentrated by living organisms (Callahan et al. 1979). Thallium(I) is more stable and resembles the alkali metal cations in many of its chemical properties. Thallium(III) forms many organic compounds (Zitko 1975); however, the toxicity of thallium(III) has been explored only lightly.

Thallium is slightly more acutely toxic to mammals than mercury. The similarity between kinetic profiles of inorganic trivalent and monovalent thallium species suggests that they are converted in vivo to one chemical form, probably monovalent thallium (Sabbioni et al. 1980). Isomorphic with potassium, thallium(I) is readily absorbed and distributed throughout the body, and can substitute for potassium and other monovalent cations in enzymatic reactions. The affinity of thallium(I) for enzymes is 10 times higher than that of potassium, which may cause the observed toxic effects (Zitko 1975). Thallium(I) uncouples oxidative phosphorylation, adversely affects protein synthesis, and inhibits a number of

enzymes including alkaline phosphatase and succinic dehydrogenase (Zitko 1975). Also toxic to plants, thallium inhibits chlorophyll formation and seed germination.

A study in the 1930s of the acute toxicity of thallium sulfate in game birds including quail (Shaw 1933) formed the basis for the TRV for these functional groups. In a study of the acute toxicity of thallium sulfate in three immature golden eagles *(Aquila chrysaetos),* the acute oral LD_{50} was estimated to be between 60 and 120 mg/kg (Bean and Hudson 1976). Using the lower end of this range as the quantified critical exposure (QCE), a TRV for raptorial birds at the INEEL was derived.

Rats exposed to thallium in their drinking water have shown effects on various neurological (Manzo et al. 1983) and reproductive (Formigli et al. 1986) endpoints. Because of the clear ecological relevance of reproductive impairment, a QCE was selected from the study of thallium-induced testicular toxicity (Formigli et al. 1986).

No information on the toxicological effects of thallium on reptiles was located.

The recommended toxicological benchmark of 1 mg/kg for thallium was used as the TRV for terrestrial plants (Will and Suter 1995).

7.3.4.16 Vanadium. Vanadium occurs naturally in igneous rock and shales, in some uranium and iron ores, and in association with fossil fuels. Vanadium has a typical native soil concentration range of 20 to 500 parts per billion (ppb). It has a natural concentration in groundwater ranging from less than 1 to 10 ppb (Dragun 1988). In the environment, vanadium is usually combined with oxygen, sodium, sulfur, or chloride (ATSDR 1992d). There is no indication that vanadium is nutritionally required by higher plants and annuals (Ammerman et al. 1973). Vanadium uptake into aboveground parts of terrestrial plants is low. However, some legumes have been identified as vanadium accumulators (ATSDR 1992d). In general, bioconcentration and biomagnification in terrestrial environments appears limited.

Most toxic effects of vanadium are associated with inhalation of vanadium pentoxide (ATSDR 1992d). Vanadium is poorly absorbed in the gastrointestinal tract, and most is excreted unabsorbed in feces (ATSDR 1992d). Ingestion of high levels of vanadium is reported to cause dehydration, emaciation, and diarrhea (Ammerman et al. 1973).

A study of vanadium toxicity in female leghorn chickens by (Kubena and Phillips 1982) was used to develop a TRV of 0.85 mg/kg/day. A TRV of 0.25 mg/kg/day was derived using a study of the effects of vanadium to mallards (White and Dieter 1978).

A study of the effects of vanadium on mice (Schroeder and Balassa 1967) was used to derive a TRV of 0.5 mg/kg-day for vanadium. Little information is available in the literature about vanadium toxicity in ruminants (Ammerman et al. 1973). A study was used to derive a TRV of 0.42 mg/kg/day (Abbey and Platonow 1968).

No toxicity information on the effects of vanadium to reptiles or invertebrates was located. The recommended screening benchmark concentration for phytotoxicity in soil for vanadium of 2 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

7.3.4.17 Zinc. Zinc is found naturally in the environment and is present in all foods (ATSDR 1993b). It is an essential element and occurs in the environment in the 2+ state. Zinc is likely to be strongly sorbed to soil. Relatively little zinc disposed of in landfills is expected to be in a soluble form. Bioconcentration factors of soil zinc by terrestrial plants, invertebrates, and mammals are 0.4, 8, and 6, respectively (ATSDR 1993b).

Excessive dietary zinc has been shown to cause copper deficiency and anemia (ATSDR 1993b). Cadmium also has resulted in the redistribution of zinc to the liver and kidney. Health effects associated with zinc exposure include anemia, liver necrosis, fetal resorption, and in extreme cases, cessation of reproduction (ATSDR 1993b).

Only one study revealed an NOAEL for chickens. A decrease in egg production was observed in chickens fed 20 mg/kg zinc sulfate in their diet. This LOAEL was converted to 12 mg/kg-day to develop a TRV (Stahl, Greger, and Cook 1990).

A rat developmental study by Schlicker and Cox (1968) was used to develop a TRV for small mammals. A study of sheep by Allen et al. (1983) revealed pathological changes in the liver and kidneys. Using these studies, a TRV was developed. A feeding study evaluating zinc oxide exposure in ferrets (Straube, Schuster, and Sinclair 1980) was used to develop a TRV for mammalian carnivores. A study using dogs by Drinker, Thompson, and Marsh (1927) was used as the basis for deriving a TRV for mammalian omnivores.

A critical concentration of 1,000 µg/g (mg/kg) zinc in food on a dry weight basis for reproduction was reported in a study on the toxicity of zinc nitrate to the isopod (*Porcellio scaber*) (Hopkin and Hames 1994). Because the bioconcentration factor for zinc for terrestrial plants is 0.4 and the maximum detected concentration in site soil is 2,400 mg/kg, the conservatively estimated plant concentration of 960 mg/kg falls below this critical concentration. Thus, while invertebrate toxicity data for soil is not available, there is some evidence that the concentrations of zinc at the site would not result in adverse effects in invertebrates at the site.

The recommended screening benchmark concentration for phytotoxicity in soil for zinc of 50 mg/kg was used as the TRV for terrestrial plants (Will and Suter 1995).

No information on the toxicological effects of zinc to reptiles was located.

7.3.5 Development of TRVs for Organic Contaminants of Potential Concern

This section contains summaries of the information used to determine the TRVs for the following organic COPCs at WAG 5:

- Aroclor-1248
- Aroclor-1254
- Benzo(a)pyrene
- Xylene.

7.3.5.1 Aroclor-1248 and Aroclor-1254 (Polychlorinated Biphenyls). Polychlorinated biphenyls are a family of compounds that vary widely in physical, chemical, and biological properties. The fate of PCBs in the environment is affected by biodegradation and photolysis. Nondestructive processes that affect the distribution and transport of PCBs biphenyls are absorption, volatilization, and bioaccumulation. Polychlorinated biphenyls tend to have high octanol-water partitioning coefficients and low water solubilities and sorb strongly to soil organic matter. The tendency of PCBs is to sorb to soil and hence their environmental persistence increases with the degree of chlorination as well as with the organic content of the soil. Polychlorinated biphenyls with a high degree of chlorination degrade very slowly in the environment. Significant leaching of PCBs from soil is not likely. The biota are another environmental compartment into which these compounds may be partitioned. Volatilization and transport as an aerosol followed by fallout with dust or rain is the probable cause of the widespread distribution of PCBs (Callahan et al. 1979).

PCBs comprise a physicochemically and toxicologically diverse group of 209 compounds whose widespread use and chemical stability have made them ubiquitous in the environment. Because of their generally low acute toxicity, effects on environmental receptors are more likely to be sublethal and chronic than acute. Toxicity and risk assessment of PCB mixtures is complicated by the fact that the 209 congeners differ markedly in both the severity and the nature of their biological effects. The toxic potency of individual congeners is dependent on their structure. While the approximate isostereomers of 2,3,7,8-tetrachlorodibenzo-p-dioxin—that is, coplanar molecules with chlorine atoms in the lateral (but not ortho) ring positions—are the most toxic (and carcinogenic in some species), many others manifest very low acute or chronic toxicity.

In the environment, PCBs occur as mixtures of congeners, but their composition differs from the commercial mixtures. After release into the environment, the compositions of PCB mixtures change over time through partitioning, chemical transformation, and preferential bioaccumulation.

PCBs can accumulate selectively in living organisms. PCBs are highly soluble in lipids and are absorbed by fish and other animals. Rates of metabolism and elimination are slow and vary by congener (Matthews and Anderson 1975). Bioaccumulation through the food chain tends to concentrate congeners of higher chlorine content, producing residues that are considerably different from the original Aroclors (Schwartz et al. 1987).

Chlorine content was formerly regarded by some scientists as correlated with cancer risk. Recently, however, Aroclor 1254 was found to be more potent than, 1260, which was only slightly more potent than 1242 (Brunner et al., 1996). Both the number and position of chlorines are important in determining the potency of PCB congeners. With respect to dioxin toxic equivalents several studies have ranked Aroclor 1254, 1248, and 1242 as more potent than 1260 (Harper et al. 1995; Safe 1994; Harris et al. 1993; Hong et al. 1993; Schulz et al. 1989). While Aroclor 1260 may be considered less toxic it does persist longer in the environment and in the body (persistence is not synonymous with toxicity; however, it is reasonable to suppose some correlation between persistence and toxicity).

Overall toxicity studies tend to rank Aroclor 1254 as the most toxic followed by Arolclor 1260, 1248, 1242, and 1016 being the least toxic and least persistent of the aforementioned congeners.

The most toxic congeners also are the most potent inducers of mixed-function oxidases as well as some Phase II enzyme activities (reviewed by Safe 1992). These enzymes metabolize not only the inducing PCBs but also a variety of endogenous molecules, such as steroid hormones, that are necessary for normal physiological function. As a result, PCBs may exert adverse effects on development and reproduction in various vertebrate species including birds (Koval, Peterle, and Harder 1987). In addition, the difference in the sensitivity of various species to the PCB compounds is considerable. Particularly sensitive species include some birds, guinea pigs, and mink (McConnell 1985).

Dahlgren and Linder (1971) and Dahlgren, Linder, and Carlson (1972) examined the effects of Aroclor-1254 exposure in pheasants. Though no NOAEL was identified in this work, its focus on a wild species and dosing of both sexes made it attractive for TRV development. Nine to 10 mg/kg-day Aroclor-1254 reduced sperm concentrations in American kestrels and Falco sparverius (Bird et al. 1983).

Linder, Baines, and Kimbrough (1974) identified NOAELs for Aroclor-1254 in a two-generation reproductive study in rats. Many studies have focused on the toxicity of various PCBs to mink, which is a sensitive species (Eisler 1986; EPA December 1993). A study by Aulerich and Ringer (1977) revealed that mink are very sensitive to these compounds and that the lethal dose varied inversely with the chlorine content of the PCBs.

In laboratory studies PCBs have been shown to decrease the survivability and hatchability of birds (Schwetz et al. 1974). Porphyria cutanea tarda, which is one of the symptoms of embryo mortality, endema, and deformities have been seen in piscivorous birds exposed to PCBs.

7.3.5.2 Benzo(a)pyrene (Polyaromatic Hydrocarbons). Polyaromatic hydrocarbons (PAHs) are ubiquitous in nature primarily because of natural processes such as forest fires, microbial and terrestrial vegetation synthesis, and volcanic activities (Eisler 1987b). The molecular weight of the individual PAHs affects their mobility and solubility in the environment, with lower-weight compounds (acenaphthene, acenaphthylene, anthracene, fluorene, phenanthrene, fluoranthene, 2-methylnaphthalene, and pyrene) being of more concern because they are generally more volatile and soluble than higher-weight compounds [benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene], which have strong sorption properties (ATSDR 1992c).

In aquatic environments, PAH partitioning in sediments occurs in an equilibrium partitioning process, with a potential for localized occurrences of high levels of dissolved PAHs (Edwards 1983). Polyaromatic hydrocarbons will adsorb strongly onto suspended particulate and biota. The ultimate fate of those that have accumulated in the sediment is believed to be biodegraded and biotransformed by benthic organisms (EPA 1980).

In general, PAHs show little tendency to biomagnify, despite their high lipid solubility, because most are rapidly metabolized by vertebrates. Plants and invertebrates, however, may bioaccumulate high concentrations of these compounds. Most studies conducted on PAHs are single compound laboratory tests that are inapplicable, for the most part, to field situations. Organism and species responses to these compounds are variable and are largely affected by the presence of other inorganic and organic compounds (Eisler 1987b). Though ecotoxicological data are scarce, the tendency is for many PAHs to be either carcinogenic (compounds of high molecular weight) or acutely toxic (compounds of low molecular weight) to many organisms. In addition, chronic toxicities, mainly seen as increased frequencies of hyperplasia and neoplasia in aquatic invertebrates, fish, and amphibians, have been demonstrated in areas with high sediment PAH concentration (Eisler 1987b).

7.3.5.3 Xylene. Acute exposure to xylene via inhalation primarily caused central nervous system effects, though acute liver injury was observed in guinea pigs given 1 to 2 g/kg-day intraperitoneally (WHO 1981). An oral LD_{50} value of 4,300 mg/kg has been reported for rats (TOXNET 1994). Chronic studies indicate that xylene has a relatively low toxicity over the long-term. No changes were found in rats, guinea pigs, dogs, and monkeys continuously exposed to 80 ppm for 127 days nor in rats exposed to 700 ppm for 130 days (WHO 1981). Ungvary et al. (1980) evaluated the toxicity of xylene in rats. Rats were exposed via inhalation to 35, 300, or 700 ppm continuously on days through 14 of gestation. No adverse effects were observed, and the authors concluded that xylene was not teratogenic. A commercial mixture of xylene was given to mice via gavage at doses of 0; 520; 1,030; 2,060; 2,580; 3,100; or 4,130 mg/kg-day on days 6 through 15 of gestation (Marks, Ledoux, and Morre 1982). No adverse effects were observed in either dams or fetuses exposed to levels of 1,030 mg/kg-day or less. An exposure of 2,060 mg/kg-day and higher approached lethal levels in dams. Fetal weight was significantly decreased, and the average percentage of malformations in fetuses significantly increased also at such dose levels.

An NOAEL of 250 mg/kg-day was developed based on a well-designed study with animals from two species: F3444N/N Rats and B6CF1 Mice. Adult males and females were tested for 103 weeks, and a comprehensive histology was performed. Toxicity reference values for mammalian receptors were developed from this study.

No data on the toxicological effects of xylene to avian, reptilian, or plant species were available.

7.3.6 Identifying Uncertainty Associated with Toxicity Reference Values

Though QCEs should be derived from the best available literature and all the uncertainties that could be reasonably accounted for are included in the AFs used to calculate TRVs, it is unlikely that any single scheme could suffice to extrapolate available toxicity data for all chemicals among all species. Thus, the remaining uncertainty in these criteria may be even greater than that associated with exposure estimation. Some of the extrapolations required in TRV development are listed in Table 7-17. Toxicity reference values are themselves dependent not only on extrapolation procedures but also on sampling adequacy and analytic accuracy, and the completeness and accuracy of response measurements in variable populations of test organisms. Combining the results from different species, gathered under different experimental conditions, and extrapolation of the results in test organisms to populations of resident species introduces additional, potentially significant sources of error as follows:

- While classical human toxicology relies on extrapolation of toxicity data from a handful of mammalian species to one species, an ecotoxicological evaluation must rely on extrapolation from a few test species to a larger number of receptor species spanning variable (and often large) ranges of phylogeny, anatomy, physiology, and life histories. Further, the spatial and temporal heterogeneity of exposure and conditions in natural systems can cause large variations in the doses and responses observed.

- Organisms in the environment are rarely (if ever) exposed to pure compounds alone, but rather to complex mixtures of chemicals for which the effects in combination are unknown.

- Chemicals may be volatilized, and transformed to more or less toxic products sequestered in the environment.

Table 7-17. Extrapolations required for developing TRVs.[a]

Extrapolation	Example
Between taxonomic groups	From laboratory mouse to field mouse
Between responses to stressor	From mortality in dogs to a no-observed-adverse-effect-level in bobcats
Between laboratory and field conditions	From cage to steppe
Between individual animals to population	From decreased growth rate in captive individuals to effects on a wild population
Between short- and long-term exposure conditions	From acute or subchronic toxicity tests to lifetime exposure
Between laboratory and natural exposure media	Percent uptake of chemical mixed with laboratory diet versus adsorbed to soil
Between spatial scales	Evaluation of the impact of exposure to a contaminated field on predators for which the foraging range is 50 times as large

a. Adapted from the Environmental Protection Agency (EPA 1992).

Our lack of knowledge of environmental variables and limited ability to replicate them in the laboratory or control them in the field results in a high level of uncertainty in our predictions of the effects of stressors on any given ecosystem component from laboratory toxicity tests.

7.4 Risk Characterization

Risk characterization is the final step of the WAG ERA process. The risk evaluation determines whether there is any indication of risk from the contaminant concentrations and the calculated dose for the INEEL functional groups, T/E species, and species of concern and considers the uncertainty inherent in the assessment. For a WAG ERA, the risk characterization step has two components starting with a description of the estimation of risk. A summary of the risk evaluation follows the risk estimation. These two components are described in the following sections.

7.4.1 Risk Estimation

Risk is estimated by comparing the calculated dose to the TRV. Exposure parameters used to calculate dose to functional groups, T/E species, and species of concern are outlined in Section 7.3.2. Soil concentration data developed for the human health risk assessment were used to calculate dose to ecological receptors at each ARA and PBF site of concern. The results of the dose calculations are presented in Appendix I. The use of chemical concentration data developed for human health risk assessment is assumed to be representative of the range of concentrations to which ecological receptors using a site at WAG 5 are likely to be exposed. The effect of uncertainty introduced from sample collection and analysis is reduced by basing risk estimates on the 95% upper confidence limit of the mean for the WAG 5 COPC concentration estimates if available. The resulting concentration estimates, used to estimate intakes, are an upper-bound estimate of the concentrations observed at the re: ied sites. This approach provides protection for ecological receptors and accounts for the uncertainty introduced by sampling, analysis, seasonality, and natural variation.

If the dose from the contaminant does not exceed its TRV (i.e., if the HQ is less than 1.0 for nonradiological contaminants and 0.1 for radiological contaminants), adverse effects to ecological receptors from exposure to that contaminant are not expected, and no further evaluation of that contaminant is required. Hence, the HQ is an indicator of potential risk. Toxicity reference values are developed in Appendix G and discussed in Section 7.3.3. Hazard quotients are calculated using the following equation:

$$HQ = \frac{Dose}{TRV} \tag{7-14}$$

where

HQ = hazard quotient (unitless)

$Dose$ = dose from all media (mg/kg/day or pCi/g/day)

TRV = toxicity reference value (mg/kg/day or pCi/g/day).

Hazard quotients are derived for all contaminants, functional groups, T/E species, and species of concern identified in WAG 5 for each site of concern. The dose estimation and HQ calculations are presented in Appendix I. A summary of the results of the HQ calculations is provided in Table 7-18.